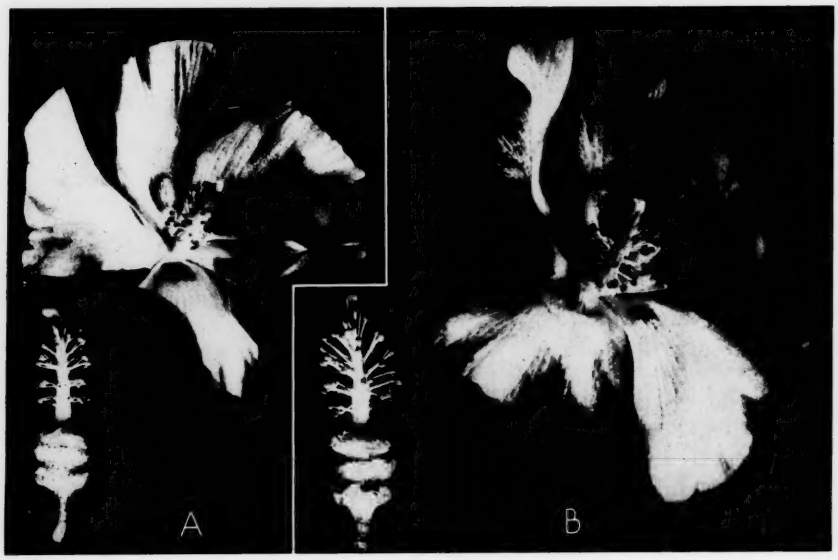
The Journal of HEREDITY



A FERTILE POLYPLOID COTTON

(SEE PAGE 39)

Diet and Cancer in Mice

Inherited Aversion to Sugar

Ring Chromosomes in Tobacco

Extra Fingers and Toes Inherited

Genetics of Root-knot Resistant Beans

Brazil's Best Families Have Most Children

Genes on Chromosomes from Different Tissues

VOLUME 31 JANUARY, 1940 NUMBER 1

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CONTENTS

THE UNIFORMITY OF THE GENE COMPLEX
By C. A. Berger 3

INHERITED INABILITY TO EAT SUGAR
By S. Davidenkov 5

CANCER OF THE MAMMARY GLAND IN MICE
By Leonell C. Strong 9

NATURAL EUGENICS IN BRAZIL
By John B. Griffing 13

INHERITANCE IN *Nicotiana tabacum*
By K. R. Stino 19

HEREDITARY POLYDACTYLY
By R. A. Hefner 25

NOTE ON WOOD'S PAPER ON PARENTAL INSTINCT
By H. W. Norton 29

FERTILITY AND CHROMOSOME LENGTH
By D. Kostoff 33

ROOT-KNOT RESISTANCE IN BEANS
By Keith C. Barrons 35

THE PRODUCTION OF POLYPLOIDS IN GOSSYPIUM
By J. O. Beasley 39

Notes and Reviews

LINKED ALBINISM AND EYE DEFECT? 4

MODERN STATISTICAL METHODS (Rev. of GOULDEN: *Methods of Statistical Analysis* and RIDER: An Introduction to *Modern Statistical Methods*)
By W. Lawson Russell 7

SYMBOLS FOR BACKCROSS AND BACKCROSS GENERATIONS
By A. R. Sidky 8

COALESCENT TWINS 16

NINETY YEARS' PROGRESS IN LIFE EXPECTANCY 18

MA-HIZ, MARISI OR MAIZE (Rev. of MANGELSDORF and REEVES, *Origin of Indian Corn*)
By G. W. Beadle 24

THE SYMBOLIZING OF GENES AND OF CHROMOSOME ABERRATIONS 27

UP THE LADDER (Rev. of SIEGEL: *Population, Race, and Eugenics*) 34

DANZIG HONORS SCIENTISTS 38

GORY DETAILS GALORE (Rev. of WIENER: *Blood Groups*)
By Clyde E. Keeler 48

STUDENT SUBSCRIPTIONS

The **Journal** has obvious advantages as a supplementary text in genetics classes. Since genetics deals mainly with form and pattern differences in organisms the **Journal** offers an unexcelled introduction to current developments, which have an added interest and appeal on account of the large number of illustrations published.

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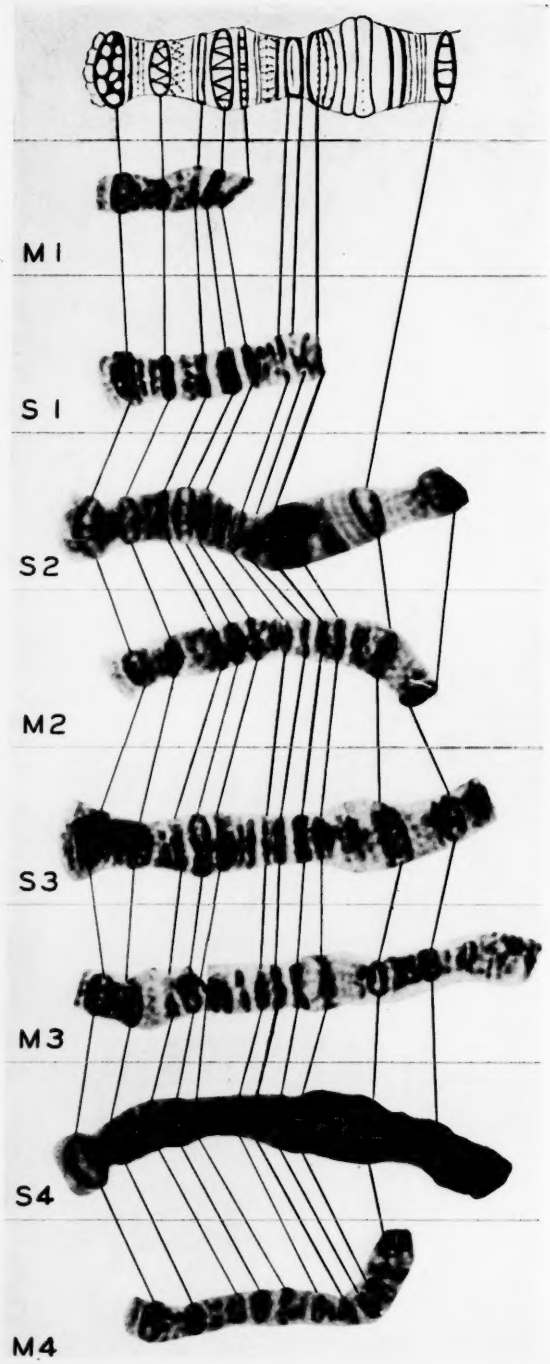

CHROMOSOMES FROM DIFFERENT TISSUES COMPARED

Frontispiece

Photomicrographs of end *1* of chromosome *A* in the midgut (*M*1, *M*2, *M*3, *M*4) and in the salivary glands (*S*1, *S*2, *S*3, *S*4) *of Sciara ocellaris.* The more prominent "landmarks" in the different specimens are connected by lines. Between these lines a correspondence in the finer bands can be observed in many places. This represents direct cytological evidence supporting the generalization that all cells in a given organism have a complete set of chromosomes and genes. All from aceto-carmine smears, × 1350.

THE UNIFORMITY OF THE GENE COMPLEX

In the Nuclei of Different Tissues

C. A. BERGER
Woodstock College, Maryland

ONE of the great generalizations of cytology is that the nuclei of the various tissues of any organism have the same chromosome content. Outstanding exceptions, such as chromatin diminution in Ascaris and Sciara, and polyploid tissues in a number of plants and animals, serve to increase rather than to lessen the validity of this generalization. One of the most reasonable and well founded assumptions of modern genetics is that this gross chromosome similarity reflects a similarity or uniformity of the gene content of different tissues. The giant Balbiani-type chromosomes of the Diptera offer an opportunity to obtain visual evidence on this important point. In the salivary glands the linear structural differentiations visible in these giant chromosomes have been shown to be correlated with the longitudinal distribution of genetic materials. Balbiani-type chromosomes occur in other tissues than the salivary glands and in a few favorable cases reach a size sufficiently large to permit of detailed examination. A comparison of the pattern exhibited by the giant chromosomes of different tissues should yield visible evidence as to whether or not the genetic materials in the chromosomes of the two tissues are the same. Such a comparative study was made of the Balbiani-type chromosomes of the salivary glands and of the mid-gut of Sciara and no differences in pattern were detected.

During the past year two investigators, Makino[4] and Frolova[2,3], made independent comparative studies of the nuclei of many different tissues is several species of Drosophila. They agree that all larval nuclei are alike in containing the haploid number of paired chromosomes all of which are united at one end in a chromocenter. It is to be noted that both of these studies dealt with gross nuclear morphology. Neither of the investigators compared the banding pattern of the chromosomes of different tissues.

The results recorded here are based on aceto-carmine "smear" preparations of old larvae of *Sciara ocellaris*. The stock was obtained from the laboratory of Dr. C. W. Metz. Some years ago[1] it was found that certain of the larval epithelial cells of the mid-gut of Sciara have giant banded chromosomes similar to those of the salivary gland cells. These cells are located in the anterior half of the mid-gut at about the level of the middle of the gastric caeca. They reach their maximum size just before pupation and only then are the chromosomes large enough for detailed study. The maximum volume is about one half that of the salivary gland chromosomes. (See Frontispiece.)

The comparative study of the banding pattern of the giant chromosomes of the mid-gut and of the salivary glands revealed no constant differences. It was possible in mid-gut preparations to identify chromosomes corresponding to those designated as *A, B, C and X* by Metz[5,8] in his salivary gland studies. All the more prominent "landmarks" could be readily found and in many cases the fainter pattern markings were seen to be similar to those of salivary gland chromosomes in texture and position. It must be remembered that even in salivary gland preparations there are variations in the appearance of the bands due no doubt to the physiological condition of the material and to the fixation. These variations, however, do not prevent an

observer familiar with the material from identifying definite regions.

The accompanying photomicrographs illustrate the uniformity found, better than verbal description. They show End *I* of Chromosome *A* (terminology of Metz) from salivary gland and mid-gut preparations. The more prominent bands are connected by lines. Between these lines a correspondence in the finer details can be observed in many places. Similar agreement is found throughout the remainder of the chromosome complex.

The differentiations forming the pattern of Balbiani-type chromosomes extend to the limit of visibility and perhaps beyond. The finer the character of the band the less certain is the evidence for uniformity in different tissues. Nevertheless, the absence of detectable differences and the agreement in all readily observable features provide visible evidence for the uniformity of the gene content of different tissues.

Summary

Giant Balbiani-type chromosomes, similar to those of the salivary glands, are found in certain mid-gut cells of old Sciara larvae. A comparative study reveals uniformity of the banding pattern in the chromosomes of these two tissues.

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◆

Linked Albinism and Eye Defect?

To the Editor:

There is a family in Ogden, Utah, which seems to demonstrate the inheritance of albinism along with a certain eye defect. Three out of about sixteen children in the family are albinos and these three have defective vision other than the extreme sensitivity to light experienced by all albinos. There is a similarity in the name of the family and the maiden name of the mother, but the children denied that there was any blood-relation between their parents. The family formerly lived in Virginia.

Should any student of genetics be interested in making a careful study of this family, I will supply an address for a starting point.

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Albinism has been frequently shown to be a simple recessive and when a community is found with a large number of albinos it probably indicates inbreeding. A study of such a group might reveal other rare recessive factors. An albino family has also been reported in Avery County, North Carolina, where several generations of the pedigree are believed to be available. These may bring linkages to light when the available data are treated by the newer statistical methods.

—Editor.

INHERITED INABILITY TO EAT SUGAR

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AMONG the varied congenital and often inherited peculiarities of taste there is one particular anomaly which consists of a *congenital incapacity to eat any kind of sweet food.* We shall further term this curious condition "aglycophagia."

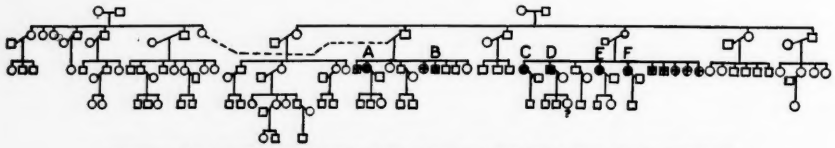
The anomaly has been observed in six members of the same family, which fails to record any intermarriages. In every other respect the members of the family are perfectly sound people, only a few of them suffer from headaches and general nervousness. Individuals affected with aglycophagia have been found to occur in two series of brothers and sisters (see Figure 1.) In the first series consisting of seven children there are two cases of aglycophagia (*A* and *B*), while in the second series of five children, as many as four are affected with the anomaly (*C, D, E* and *F*). *C* is affected to a somewhat milder degree. The parents, as well as all of the children of those affected with aglycophagia can readily eat food of any taste. In all the cases the anomaly revealed itself in early childhood, and apparently has failed to change in the course of the individual's life.

The propositus *B*, (Figure 1), is 26 years old. He cannot remember having ever been able to eat any food that tasted sweet. His mother remembers having noticed that as a very small child he used to cry when he was made to taste a sweet bun. He cannot eat sugar, honey, jam, fancy cakes, candies, chocolate, ice-cream or anything of that kind. Nor can he eat any fruits or berries, with the exception of cranberries and unripe red bilberries. He cannot eat any kind of cake, or take any alcoholic drinks, or even mineral water. He cannot use saccharin in his food. His diet mostly consists of rye bread, unsweetened wheat bread, uncooked milk and other dairy products. He can eat potatoes, unless they have been frozen, which gives them a slightly sweet taste, so that he can no longer eat them. His "ideal of tasty food" is: tea with milk and no sugar and a slice of rye bread with a cucumber pickled in brine. At the same time he definitely states that he does not object to the taste of anything sweet. On the contrary, he sometimes even takes a candy or a lump of sugar into his mouth and sucks it for a moment. He invariably spits it out, however, and then rinses his mouth. He can never make himself swallow anything sweet as "it is as bad as taking the most repulsive kind of medicine." Several times, when there was nothing else to be had, he had forced himself to eat sweet food, which invariably resulted in nausea and a most unpleasant physical weakness; he also had a feeling of there being something "abnormal in the whole of his body," felt "apathetic," and "got tired easily." He states that he was not able to run quickly or even clench his fists with his usual strength. He had a feeling "as though he wanted to wriggle and writhe."

In every other respect he is a perfectly healthy man, very capable and active, physically strong, but slightly nervous. From time to time he suffers from eczema which affects his arms and legs, but this is no way connected with his having eaten anything sweet.

He consented to enter the clinic for a short time to undergo a more thorough examination, which failed to reveal any deviations from the normal standards either of the nervous system or the viscera. His blood contained 36.9 mg. p.c.K, and 10.0 mg. p.c. Ca. The amount of diastase contained in blood and the saliva was normal, but that contained in the urine was reduced one half.

The subject under observation was



FAMILIAL INHERITANCE OF AN INABILITY TO EAT SUGAR

Figure 1

Pedigree showing the two groups of cousins who were unable to eat sugar in any form. The one individual (*B*) who underwent extensive clinical tests became flushed, feverish, and complained of pain in his stomach when given sugar at the clinic. His conception of the perfect meal was tea without sugar, rye bread and brine pickles. The affected individuals lived mostly on rye bread and dairy products. The crossed symbols indicate death in infancy.

given 50.0 c.c. of cane sugar and this brought on a very severe condition: his face became flushed; he felt feverish, and suffered from suffocation and complained of pressure in the region of the pit of the stomach. His pulse became arythmic, the chronaxia of the flexors and of the extensors showed a manifest tendency towards levelling (0.12 σ and 0.16 σ instead of his usual 0.12 σ and 0.2 σ; the sugar-curve of the blood became atypical and gave a tardy rise. Before the intake of sugar the sugar content amounted to 94 mg. p.c.; in half an hour it was 102; in an hour 98; in an hour and a half 94; and in two hours it rose to 176 mg. p.c.). Four hours after the test the patient's usual condition was restored, chronaxia gradually became normal (0.09 σ and 0.2 σ), but for a long time after that he could not think of sugar without a feeling of repulsion, so that he even threw away the lumps of sugar which remained on his night table and refused point blank to undergo any further experiments of this kind.

According to the patient's statements, his eldest sister, *A*, was characterized by the same anomaly as himself.

His cousin, *F*, whom we had occasion to observe, had exactly the same diet as our patient to the extent that she was likewise unable to eat potatoes that had been frozen because of their being slightly sweet. If she ate anything sweet, for instance, a tiny piece of chocolate, she "felt unwell," had nausea and to avoid this unpleasant feeling had to eat a piece of rye bread with salt and drink some water. She could not say what might happen to her if she ate a larger quantity of anything sweet, for she had never been able to make herself do so: "better starve than eat a sweet bun" she said. Contrary to her cousin, the propositus, she dislikes the taste of anything sweet, and though she thinks the smell of honey pleasant, she says she "could never think of eating any." Her teeth, as well as those of her cousin, are marvelously sound and intact.

Her elder brother *D* has the same peculiarities, though he can drink beer and lemon-vodka; he tried to eat saccharin but could not swallow it. Her sister *E*, who suffers from eczema, has exactly the same peculiarities as regards anything sweet as *B* and *F*. Her eldest sister *C* has the anomaly in a somewhat milder form; she is able to take a small quantity of tea with a little sugar, as well as to eat a slightly sweetened bun and take alcoholic drinks. But she cannot eat honey, fruit, jam or sweet dishes any more than the others.

The inner mechanism of this peculiar anomaly does not seem to be perfectly clear. The fact that those affected with it are unable to take saccharin seems to indicate that it is not so much the incapacity for eating substances of a definite chemical structure as the impossibility of eating anything which tastes sweet that we have to deal with in these cases, unless we assume that the incapacity of these individuals to eat mono- and disaccharids which is due to the chemical properties of the latter may have spread to any substance that tastes sweet on the principle of the conditioned reflex. Certain bio-chemical anomalies,

such as a reduced quantity of diastase in the urine or an atypical sugar-curve, might, apparently, be regarded as having resulted from the peculiarities of a continued absence of sugar from the diet. The capacity for eating polysaccharids which do not taste sweet and which are then converted into glucose, likewise seems to indicate that the anomaly is not due to disturbed carbohydrate exchange. Thus, we are led to believe that it is the taste of anything sweet which is the incitant of the pathologic condition. The symptoms of the latter hardly give sufficient grounds to regard the entire syndrome as a kind of allergy. The syndrome is highly peculiar and seems to rank closest to family myoplegia, the atypical rudimentary forms of which are well known to us, as well as its connection with this or the other form of diet. In this respect it is of interest to note the statement made by the subject of our observations as regards his incapacity for running fast or clenching his fists with strength after he has eaten something sweet, as well as the objective changes observed in his motor chronaxia following the intake of sugar.

The above described syndrome does not wholly resemble any of the clinical affections so far known to us and, therefore, we have thought it fit to publish the results of our observations.

✦

MODERN STATISTICAL METHODS

THE methods described in these two books are based on "Student's" distribution, the chi-square distribution, the analysis of variance and the design of experiments, methods originated or developed by R. A. Fisher. Since the publication of Fisher's *Statistical Methods for Research Workers* and *The Design of Experiments* several books have appeared which give straightforward descriptions of Fisher's methods. New books in this field will arouse interest, therefore, primarily in the manner in which insight into the principles or application of these methods is presented.

Goulden's book* is designed for research workers in agriculture and emphasis is placed upon application rather than upon theory. Forty per cent of the text is devoted to 48 examples and 64 exercises, the examples occupying three-fourths of this space. In accordance with the author's view "that training in facility is necessary in order that statistical computations may be attacked with determination and completed in a reasonable length of time" the examples and exercises were selected for adequate length and not for simplicity. An elementary knowledge of statistical theory is held to be a prerequisite, but the author reviews basic concepts briefly and discusses principles of experimental design. Though undoubtedly helpful as a whole this part is, perhaps because of its necessary condensation, the weakest. For example, it is misleading to give, as sole reason for the adoption of the standard deviation as a measure of variability, the fact that the mean deviation, taking sign into account, is zero. Goulden gives tables of t, x^2 and Snedecor's table of F $(= e^{2z})$.

Rider's book† is designed both for beginners and for those who wish to understand modern methods. Emphasis is placed upon theory and mathematical derivations of formulae are given. In accordance with the author's view that underlying principles should not be "lost sight of in a maze of arithmetical computations" examples were selected for simplicity. There are, however, 92 exercises. It may be felt that development of the-

*Goulden, C. H. Methods of Statistical Analysis. 277 pp. $3.50. Wiley, New York. 1939.
†Rider, P. R. An Introduction to Modern Statistical Methods. 220 pp. $2.75. Wiley, New York. 1939.

ory starts too frequently at an intermediate point rather than from basic concepts and that, in general, the mathematics is not accompanied by sufficient verbal explanation. This would not necessarily be a criticism if the author had not claimed to be offering a first course in statistics. Rider gives tables of the probabilities and ordinates of the normal curve and Fisher's tables of t, x^2 and z.

So far as the subject matter of both books is concerned, a treatment of modern statistical methods which is confined to the methods developed by Fisher will give an unbalanced picture. Thus, both Goulden and Rider devote considerable space to regression and correlation, but make no mention of Wright's method (described by Haldane as a "very powerful method") of path coefficients.

To sum up, it is the reviewer's opinion that neither of the above books will serve alone as a complete introduction to modern methods, but that each admirably fills a real need in class work, Goulden to give details of application and computation and Rider to give mathematical theory, neither of which can be absorbed efficiently from the blackboard.

W. Lawson Russell

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✦

SYMBOLS FOR BACKCROSS AND BACKCROSS GENERATIONS

THE need for symbols to express the backcross—male and female—and the backcross generations of each, in an unambiguous and easy form is so often experienced by geneticists that it is thought that the following suggestions may be found useful by many:—

FOR USE WITHOUT SPECIAL TYPE.	SUGGESTIONS FOR SPECIAL TYPES.	
δbc	bc^{δ}	Backcross of male.
$♀bc$	$bc_{♀}$	Backcross of female.
$F\,\delta bc$	$F\,bc^{\delta}$	Offspring from backcross of male and female respectively.
$F\,♀bc$	$F\,bc_{♀}$	
$F_{1,2,3\ldots}\,\delta bc$	$F_{1,2,3\ldots}\,bc^{\delta}$	First, second, third, etc., generation offspring from backcrosses of male and female respectively.
$F_{1,2,3\ldots}\,♀bc$	$F_{1,2,3\ldots}\,bc_{♀}$	
$\delta bc_{2,3\ldots}$	$bc^{\delta}{}_{2,3\ldots}$	Second, third, etc., backcrosses of male and female respectively.
$♀bc_{2,3\ldots}$	$bc_{♀2,3\ldots}$	
$F_2\,\delta bc_3$	$F_2\,bc^{\delta}{}_3$	Example: — Second generation offspring from third backcrosses of male and female respectively.
$F_2\,♀bc_3$	$F_2\,bc_{♀3}$	

A. R. Sidky.

Institute of Animal Genetics,
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CANCER OF THE MAMMARY GLAND IN MICE

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EVIDENCE that cancer of the mammary gland may originate by a process of genetic change comparable to somatic mutation has been published.[15,16] This conclusion has been verified by Bittner[3,4] and by Cloudman.[6,7] More recently Dunning, Curtis and Bullock have expressed a similar idea for the origin of tumors induced by parasites and chemical irritants.[8] The establishment of pure strain mice that give rise to spontaneous tumors, especially of mammary tissue, has led geneticists to hope that the occurrence of these neoplasms would be placed on a mendelian basis. Such a conclusion, for the origin of spontaneous tumors, derived from actual data, has, however, not been realized. Many interpretations have been forthcoming. The simplest, that cancer in mice is determined by a single mendelian recessive,[19] advocated for so many years by Maud Slye, has very recently been greatly modified. Her original conception was that "In every one of these five thousand neoplasms, both external and internal, the inheritance behavior of cancer susceptibility has been that of a simple mendelian recessive." It is her latest opinion that, in cancer susceptibility, different types of tumors are due to separate or distinctive mendelian recessive genes.[20] In a recent paper she advocates that "malignancy is transmitted as a localized recessive character, each type of malignancy being a unit character and capable of suppression by a dominant. That is, one unit recessive character for carcinoma, one unit recessive character for sarcoma, and one unit recessive character for leukemic disease." Another simple interpretation is that of Clara Lynch, who maintained that cancer of the lung in mice was inherited as a simple mendelian dominant. There is a similar mechanism, according to Lynch, involved in the induction of lung tumors by the carcinogens.[12] Her recent stand may be taken from her paper presented at the Symposium on Cancer at Atlantic City, December, 1936. "(1) External agents cause cancer in mice, (2) the response to the agent is controlled by the constitution of the individual, (3) constitutional differences in susceptibility are inherited and (4) susceptibility is apparently organ specific." Andervont[1] has verified the general conclusions as expressed by Lynch. He also concludes that "the appearance of induced tumors in the lungs of strain *A* mice or in the lungs of their offspring implied that these growths can be evoked more frequently in those mice which possess a tendency to develop them spontaneously." Bauer has maintained that cancer susceptibility was due to the inheritance of two genes, one a general cancer character, the other a specific determiner for each special type of tumor.[2] His own words were, "wir haben die Annahme von zwei eine konstitutionelle Krebsdisposition bedingenden Faktoren im Vorangehenden begrundet: Eine allgemeine Blastomanlage, welche für die Krebsfähigkeit ihres Trägers ganz allgemein erforderlich ist, und eine bestimmte Organdisposition, welche die Lokalisation bei gegebener Krebsfähigkeit determiniert." It has also been maintained that cancer susceptibility is determined by the inheritance of multiple factors. Among

*This experiment has been made possible by grants from the Anna Fuller Foundation, the International Cancer Research Foundation and the Jane Coffin Childs Memorial Fund for Medical Research.

the early attempts to determine the nature of susceptibility to spontaneous tumors by the use of hybridization experiments involving pure strain mice may be mentioned those by Little.[10] He stated, "The incidence of tumors in the F_1 and F_2 generation is indicative of genetic complexity." He also maintained that "the variability of type and site of neoplasm in F_2 suggests that modifying factors may be determining the location of certain of the tumors."

Crosses Inconclusive

Hybridization experiments, based upon the use of highly inbred strains of mice have not produced conclusive evidence for the analysis of the mechanism involved in cancer susceptibility. Cancer susceptibility, in many outcross experiments, has behaved as a dominant occurring in high frequency of the F_1 population. The backcross generation, produced by a cross between an F_1 individual and the theoretical recessive parent, does not give a ratio of susceptible-to-cancer to resistant-to-cancer individuals that is mathematically consistent with the ratio obtained in the F_2 population.[18] This discrepancy was obtained with any combination of mendelian factors assumed to explain the situation of susceptibility and resistance in F_2 individuals.

The problem of susceptibility to spontaneous tumors in mice is more than the simple inheritance of genetic determiners as was clearly indicated by Strong in case of (a) melanoma, (b) small round cell sarcoma, and (c) leukemic lymphoblastoma.[17] He concluded that these types were clearly non-genetic in their origin, since they did not occur in the lineal descendents of the mice which originally developed them. The "non-genetic" origin of spontaneous tumors in mice has very recently been greatly extended under the terms of extrachromosomal or cytoplasmic inheritance, etc.

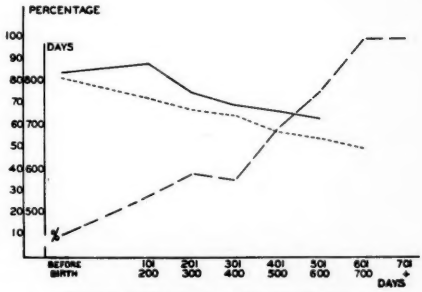
That the female parent has a more pronounced effect on the incidence of cancer in the F_1 progeny than the male parent is well known. Several investigators have published data leading to this conclusion. Among these are the collaborators of C. C. Little at the Roscoe B. Jackson Memorial Laboratory,[11] and Korteweg.[9] Korteweg concludes that, "De dispositie voor kanker van de borstklier bij bastaards tusschen twee bepaadle muizenstammen wordt door extrachromosomale zoowel als chromosomale factoren bepaald." Lynch,[13] on the other hand, has concluded that maternal inheritance does not seem to play an important role, if any, upon the incidence of tar-induced cancer of the lung in mice. MacDowell has also indicated a differential maternal influence on the occurrence of mouse leukemia.[14] He states that, "It may be added that when the leukemic heredity is brought into the hybrids by mothers the incidence in both first generation hybrids and backcross is again intermediate between the parental generations, but in both hybrid generations the incidence is significantly higher when the mother brings in the leukemic heredity." Little expressed the opinion that this phenomenon of maternal inheritance indicated cytoplasmic inheritance, or to use his own expression, "direct transmission through the extrachromosomal portion of the germ cell." Bittner, on the other hand, taking into consideration the results of fostering susceptible-to-cancer young on immune-to-cancer mothers and finding that spontaneous cancer did not eventually ensue, maintains that the "milk factor" eliminates the possibility of "cytoplasmic or extrachromosomal inheritance."[5]

Effect of Diet

The ease by which the age distribution of cancer of the mammary gland in mice may be influenced by changes in the diet upon which the individuals have keen kept, precludes the conception that cancer susceptibility is inherited by any simple mechanism. For example, when female mice of the *CBA* strain, which have been used for breeders, are kept on an oatmeal mixture, less than five per cent of them develop spontaneous tumors; when *CBA* mice are kept on a commercial diet (Diet A) from 75 to 80 per cent of them develop spontaneous

tumors. Obviously before proper evaluation of data purporting to analyze the inherited mechanism for cancer susceptibility can be had, the influence of diet and perhaps other environmental influences must be determined.

In this paper, data are presented on the incidence of spontaneous tumors of the mammary gland in 896 female mice of the *CBA* strain, which had been transferred from one commercial mouse diet (Diet *A*) to another commercial diet (Diet *B*) at different ages. For purposes of analysis, eight age classes depending on the time of transfer were used. The first class was made up of those individuals that were placed on Diet *B* "before birth," that is the mother was placed on Diet *B* while pregnant and the young were continued throughout life on Diet *B*. Two hundred and forty individuals were produced under these conditions, 24 of these developed spontaneous carcinoma of the mammary gland at an average age of 810.5 days; 216 died without the presence of tumors at an average age of 822.9 days. The other age classes were (2) 101-200 days of life, (3) 201-300, (4) 301-400, (5) 401.500, (6) 501-600, (7) 601-700 and (8) 701-800. Data on (a) the number of individuals in each class, (b) the number of each class developing spontaneous tumors of the mammary gland, (c) average age of spontaneous tumors, (d) the number of mice dying of some other cause than cancer, (e) the average age of death of those mice dying of some other cause than cancer and (f) the percentage of individuals developing cancer are given in Table I. Data on (a) the age of transfer from one diet to the other, (b) the average age of spontaneous tumors and (c) the average age of death for those mice dying of some other cause than cancer are given graphically on Figure 2.



DIET CHANGE AND TUMOR DEVELOPMENT

Figure 2

This chart shows graphically some of the data presented in Table I. The horizontal axis shows the age at which the mice studied were transferred from diet *A* to diet *B*. The dash curve shows the increasing percentage of mice dying of tumor as they are continued for longer periods on diet *A*. The solid curve shows the age at death (in days) of mice dying of causes other than tumor, and the dotted line shows the ages at death of mice dying of tumor.

General Discussion

The ease by which the age distribution of spontaneous tumors of the mammary gland in mice may be influenced by diet, as indicated by the data in this paper, would certainly indicate that cancer is not a "unit character." A classification of cancer and non-cancer that has been used for a genetic analysis of susceptibility and resistance to cancer in a hybridization experiment is, therefore, probably not justified. The "diet factor" that influences cancer in mice is not a temporary one (such as the "milk factor" of Bittner, said to be effective in preventing cancer in a "genetically determined" susceptible individual if the new-born individual be subjected to the milk of a foster mother for less than 48 hours) but a continuous one. Thus it exerts a continuous influence upon the susceptible-to-cancer individual before birth and, in fact, during the entire life span. Susceptibility and resistance to cancer then are not entirely genetically determined.

TABLE I. Development of Spontaneous Tumors in a CBA strain of mice.

Age of Transfer	Number of mice	Tu	Age Tu	Non Tu	Age of death non-tumor	Per cent Tu
Before birth	240	24	810.4	216	822.9	10.0
101-200	284	91	766.8	253	844.2	89.0
201-300	132	51	741.7	81	772.6	38.6
301-400	110	40	732.1	70	750.3	36.3
401-500	42	25	698.8	17	741.2	59.5
501-600	40	30	670.7	10	7...7	75.0
601-700	4	4	650.0	0	---	100.
701+	4	4	715.8	0	---	100.
Total	896	279		617		31.1

Some tendency that leads toward malignancy is received by the individual from its germ plasm. This tendency is continuously being influenced by environmental agencies especially from the diet and perhaps by other agencies not yet known or indicated.

In order to explain certain phenomena of tissue specificity, genetic susceptibility and other aspects of cancer, it is convenient to maintain that cancer is the resultant of a peculiar constitution of the individual which gives rise to malignancy. This conception is valid if one assumes that by the constitution one accepts not only the specific contribution from the germ plasm but also all the influences from the environment that have exerted an influence on that individual up to the time that malignancy occurs.

Cancer of the mammary gland in mice is not exclusively a genetic phenomenon: it is not entirely a congenital disease, nor is it solely an acquired one. It is the resultant of many factors that exert an influence over a relatively long period in the development of the susceptible-to-cancer individual.

Conclusions

1. The age distribution and the percentage of individuals showing spontaneous carcinomas of mammary tissue is significantly influenced by the diet upon which the mice have been kept.

2. Cancer of the mammary gland in mice is determined by an original contribution from the germ plasm which has been continuously influenced by factors from the diet (before birth and up to the time that malignancy shows itself).

3. Cancer is consequently not a "unit character" and a classification of cancer and non-cancer individuals in a hybridization experiment is probably not justified.

4. Further work on the nature of the susceptible-to-cancer and the resistant-to-cancer states is indicated before a final analysis on the part played by genetic determiners in these states may be made possible.

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NATURAL EUGENICS IN BRAZIL

JOHN B. GRIFFING

An article by Dr. Griffing on differential birth rates in China, published fourteen years ago in the JOURNAL, *was one of the first contributions which clearly demonstrated a different reproductive pattern from that revealed by statistical studies of European and American populations. Now he finds in Brazil a reproductive pattern quite different from either the Chinese or the North American, and one which may closely resemble that found in North America in colonial times.*

The situation is of considerable interest not only because it reveals a different pattern, but because it might also throw light on the perplexing problem of social superiority and genetic superiority. The test of producing children capable of being selected as college material involves the interaction of genetic and cultural factors whose limits have not been determined. In the pioneer economy of Brazil this matter might be studied more successfully than in the more highly specialized — and perhaps more rapidly changing—economy such as we enjoy (or suffer from) in North America.—EDITOR.

ONE who spends sufficient time in Brazil to see something besides the armadillos, dillowing in their armor, and becomes acquainted with the Brazilian people, is inevitably impressed by the size of their families. On the twenty-fifth of May, 1939, a feature article appeared in the *Folha de Minas*, daily newspaper of Bello Horizonte, capital of the state of Minas Geraes, describing the family of Senhor Modesto Barros. This news item stated that during a period of married life with one wife, of 33 years, 11 months, and 13 days, the union was blessed by 33 children. All were single, normal births, 19 being boys, and 14 girls. While such a family made news in Minas Geraes, 12 or 14 children would be considered no more unusual than such a flock would have been to one of our colonial ancestors.

While serving as director of the Agricultural College of the State of Minas Geraes the writer considered it worth while to investigate the rate of increase in superior elements of the population as represented by the families from which the students of the college came. This criterion for superiority is frequently used by students of population in the United States, but there are reasons why it may be applied even more legitimately in Brazil. (1) There, only the select few ever enter college. No democratization of education for the masses swamps the higher institutions. (2) Entrance to college is safeguarded by rigid examinations, regardless of credits. (3) Students in Brazil are, as a rule, sons of leading citizens, proprietors of extensive plantations, or of merchants, professional men, or officials. (4) Minas, and all of Brazil for that matter, is still a pioneer country, and as such, an effective sifting ground. Achievement and recognition tend to be based upon native ability and initiative. A professor from the United States gave as his observation after seven years on the college faculty: "The Brazilian students suffer the handicap of a preparation much inferior to that of students in the United States, but are a much more highly selected group, and represent more nearly the cream of the country."

For purpose of contrast data were obtained from laborers on the college campus as well as from students, and from individuals of the lower economic levels of the community, both town and country. Data from both students and laborers were carefully taken through individual personal interviews. Students were asked to give information both about their own brothers and sisters and those of their fathers and mothers. Workmen were questioned simply about their own children. Whenever answers showed lack of certainty they were discarded, as

were records of families involving more than one marriage. The records as compiled included: (1) children living, and children deceased of the groups into which the students were born; (2) children living of the groups into which the parents were born; and (3) children living and children deceased of the laboring classes. Since parents of students were already of mature years, and since some brothers and sisters of students had died after reaching maturity, those who had survived to the age of at least 21 years were counted as living. The records of the laborers were divided into complete and incomplete families according as the wife had or had not passed the child-bearing age.

The number of student families entering into the study was 285, a sample which is sufficiently large to give significance to the conclusions. Of these 166 were from the state of Minas Geraes, 86 from other states, and 33 from the city of Rio de Janeiro. Data from these different sources show certain definite trends and will be presented separately. Of the laborers, 107 families were studied, 54 complete, and 53 incomplete.

A summary of the data obtained from students is given in Table I.

The average of all cases does not of course represent a cross section of all Brazil, since by the nature of the location of the college the numbers from Minas predominate. Minas is said to be the most typically Brazilian of all the states. Settled by a gold rush which began nearly 250 years ago, the amalgamation of the European, Indian, and Negro races is there more complete than in other parts of the country. Minas also leads all the states in Brazil in total population, having something more than 8,000,000. One reason for this lead is evident when we see that her superior families average 7.61 living children. The average for the other states is 5.99, and for the city of Rio de Janeiro is smallest with 4.49. But since three children per family is adequate to maintain a group, even in Rio the superior stock is producing fifty per cent more than the number needed to keep its number constant. It is significant to note that Rio with its better developed health service and facilities for medical attention, has a much lower death rate than the interior.

In studying rural families as contrast-

TABLE I. Average number of children per family into which students were born.

Where born	Cases	Living children	Deceased children
State of Minas	166	7.61 ± .16	1.74 ± .11
Other States	86	5.99 ± .22	1.44 ± .14
City of Rio	33	4.49 ± .29	.50 ± .12
TOTAL	285	6.76 ± .13	1.48 ± .16

TABLE II. Relative size of rural and urban families of students.

	Rural			Urban		
Where born	Cases	Living children	Deceased children	Cases	Living children	Deceased children
Minas	80	8.58 ± .22	2.06 ± .18	86	6.72 ± .19	1.45 ± .15
Other states	37	6.30 ± .30	1.19 ± .17	49	5.76 ± .40	1.38 ± .21
City of Rio				33	4.49 ± .29	.50 ± .12
TOTAL	117	7.85 ± .18	1.79 ± .13	168	6.00 ± .15	1.26 ± .10

TABLE III. Trend in size of family through one generation.

	Students' parents		Parents' parents	
Where born	Cases	Living children	Cases	Living children
Minas	166	7.61 ± .16	274	7.18 ± .14
Rural Minas	80	8.58 ± .22	129	7.91 ± .21
Urban Minas	86	6.72 ± .19	145	6.59 ± .11
Other states, rural	37	6.30 ± .30	71	5.82 ± .28
Other states, urban	49	5.76 ± .40	77	6.09 ± .26
City of Rio	33	4.49 ± .29	48	5.75 ± .20
TOTAL	285	6.76 ± .13	470	6.66 ± .10

ed with urban we discover a size of family that is really remarkable. The fact should be kept in mind in reviewing these figures that they do not represent average rural population. They are families of planters. The planter, or "fazendeiro" as he is called, is the basic unit of society in that agricultural nation. He is the land owner with an estate numbering its acres by thousands. He is the counterpart of the planter of the old South in the United States. Unlike the absentee landlords of Argentina, he lives on his land and loves country life. Since Brazil has more fertile land than has the United States, only four per cent of which is yet cultivated, it will be the land of the fazendeiro for a long time to come.

Here we find on the plantations of Minas the astounding number of 8.58 living children and 2.06 dead. Rural families in general average 7.85 living as compared with 6.0 in cities, but it will be noticed that in Minas even the city families average larger than the rural of other states.

A previous study made in the United States showed that small families of superior stocks are a phenomenon of the present generation. A generation ago all families tended to have approximately five children. But today the homes from which our most brilliant children come average around three, while the families producing inferior children still have five or more.[2] It is of interest now to discover the trend in Brazil. Table III compares the number of children born to the students' parents with the number born in the previous generation.

Here we see that in Brazil, both rural and urban areas of Minas, and rural areas of other states show even larger families in the present generation than in the one preceding. Cities in other states show a slight trend toward smaller families, and in Rio such a trend is quite definite.

In Table IV we are able to compare the number of children in upper and lower classes, as represented by families from which students come on the one hand, and those of day laborers on the other. In a previous study of a similar nature made in China, the findings, published in this JOURNAL,[3] were somewhat similar to those in Brazil, and so are included in this table.

Two significant facts stand out in the table. First, for both Brazil and China the number of living children in upper-class families is substantially larger than in families of the lower classes. This is exactly opposite to the trend in the United States and in some European countries. Second, the greater contribution of superior stocks to the population is due to the terrific death rate among the lower classes. It is interesting to note that in both levels the Brazilians are more prolific than the Chinese.

From the 53 families of Brazilian laborers whose families were still incomplete it was possible to obtain an approximation of the rate of fecundity of the mothers in these families. Data from the study in China already mentioned are available for comparison.

TABLE IV. Comparison of size of family in superior and lower classes for groups in Brazil and China.

Where born	Class	Cases	Living children	Deceased children
Total for Brazil	Student	285	6.76 ± .13	1.48 ± .16
Minas, rural	Student	80	8.52 ± .22	2.06 ± .18
Minas	Laborers	54	4.85 ± .20	3.59 ± .20
China	Student	75	4.24 ± .20	1.85 ± .13
Rural China	Peasants	220	2.99 ± .08	2.33 ± .09

TABLE V. Comparison of fecundity of mothers in laborers' families in Brazil with those in like classes in China.

Locality	Cases	Average age	Age at marriage	Years married	Living children	Deceased children
Minas	53	30.7	19.7	10.9	3.70 ± .18	2.02 ± .16
China	157	28.3	18.4	9.9	1.58 ± .08	1.16 ± .08

Table V compares the size of incomplete families from a group of lower class Chinese with those of Brazilian laborers. It will be noted that the Chinese women, averaging slightly over 28 years in age already had an average of 2.74 births each, with 1.58 children living. Buck, who has made extensive studies of sample populations in China, holds that Chinese mothers have the highest rate of fecundity of the mothers of any country.[1] But even allowing for one more year of married life on the part of the Brazilians, their families still outnumber those of the Chinese two to one. Since early marriage is conducive to large families, it is interesting to note that the average age of the Brazilian brides was 19.7 years. Chinese brides, however, averaged 1.3 years younger. In compiling the data it was observed that not one of the 53 incomplete families of Minas was childless. Of the 54 complete families of laborers, only one was childless.

The rapid reproduction rate of the Brazilian people in general is reflected in the growth of population in the country as a whole. This increase is unparalleled for a nation of this magnitude in the same period. In 1900 the population of Brazil was 17,000,000; in 1920, 30,000,000; and at present, approximately 48,000,000. Immigration, of course accounts for part of the increase, though since 1930 immigration has been sharply curtailed.

Summary

1. Superior families in Brazil, as represented by those into which students are born, are noteworthy in size, being substantially larger than families of similar groups in the United States.

2. The families in the typically Brazilian state of Minas Geraes are larger than those of other states.

3. Superior families of the present generation, with the exception of those in the large cities, are larger than in the previous generation.

4. The number of living children of the planter in the state of Minas Geraes is nearly double that of the common laborer. The chief cause of this difference is the higher mortality rate of children in the poorer class.

5. A favorable differential in increase of superior over inferior classes exists in Brazil as in China.

6. The rate of fecundity of mothers in Brazil is higher than that in China.

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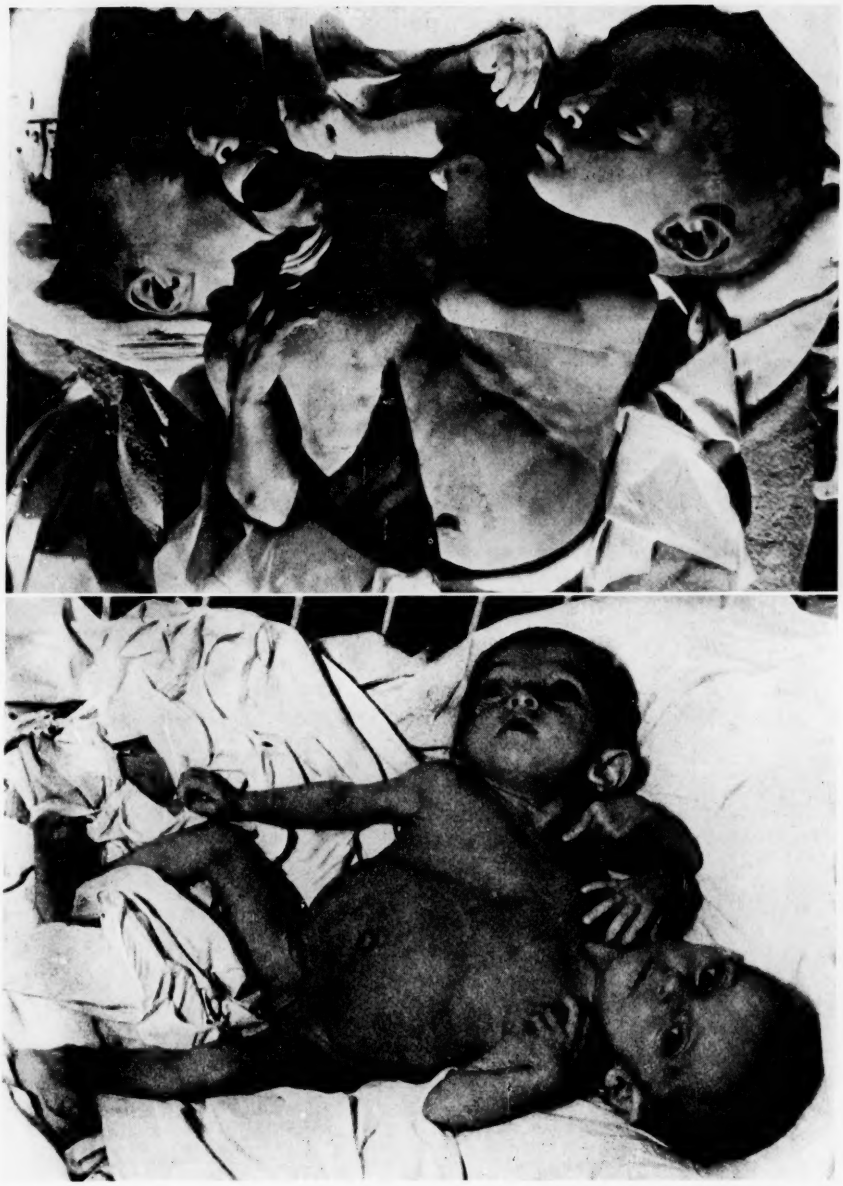
COALESCENT TWINS

DURING the past year an autopsy has been in progress on the body of the coalescent twins, Galya and Ira, who lived to be a year old and died from pneumonia while under observation at the All-Union Institute of Medicine in Moscow.

There are very few medical records of such a pair of twins having been born alive, and therefore few such twins who could be studied for a whole year by physicians and the result of these studies verified by an autopsy. The conclusions arrived at are very valuable to science, especially the discovery that the origin of sleep is not connected with the blood stream. This was proved by the fact that one twin would sleep while the other lay awake. It was realized that the explanation of this was that the twins had two separate central nervous systems, so that in spite of a common blood



SEPARATE NERVOUS SYSTEMS MADE COALESCENT TWINS ACT AS DISTINCT INDIVIDUALS

Figure 3

Photographs of the coalescent twins, Galya and Ira, shown in their crib at the All-union Institute of Medicine in Moscow. Despite their "attachment" for each other, they were rugged individualists as far as nervous upsets were concerned, as is shown in the top photograph where Galya is angrily crying and Ira is undisturbed. The twins were born in 1937 and lived to be a year old.

stream they were two separate beings.

The twins had two heads and four arms. Fusion of the bodies began at the shoulders and they had one torso and two legs. They also had a rudimentary tail three or four centimeters long.

There were two breast glands; two hearts enclosed in one membrane; four lungs; one navel formed from two umbilical arteries; two stomachs; one large liver; two sets of intestines merging into one large intestine leading to a common outlet. There were four kidneys leading to a single bladder.

Although in outward appearance the twins were the same, the autopsy (and the circumstances of their death) showed that Ira (the left hand twin, who is to the right in Figure 3) was the master-twin. Her lungs and heart were normal, while Galya's were cramped. Ira's stomach was normal while Galya's was squeezed toward the back and was pressed against by the liver.

These conditions were borne out by the fact that Galya was the first to fall ill with inflammation of the lungs, and for a time Ira's lungs and heart did extra duty for the two, keeping Galya alive longer than would have been expected.

The twins had two separate spinal columns, hence separate nervous systems. They not only slept separately, but they had individual reactions to pain and responded individually when called by name.

During their one year of life the twins were in the care of Doctors P. Anokhin and G. N. Speransky of the All-Union Institute of Medicine in Moscow. After their death these physicians continued their study of the twins and they are soon to publish a volume giving to the scientific world the facts and conclusions they have drawn from this unique study.

HELEN BLACK

✦

Ninety Years' Progress in Life Expectancy

The *Statistical Bulletin* of the Metropolitan Life Insurance Company points out that between the date of the first American international exposition in 1853 and those of New York and San Francisco in 1939 the average length of life in the United States has increased by almost a quarter of a century. About 1853 the expectancy of life for white males was only about 38 years; at the time of the Centennial Exposition, 1876, this figure had increased to almost 42 years. Rather peculiarly, there was no striking improvement during the next seventeen years, though these were the years during which fundamental discoveries were made in bacteriology and immunology by such men as Pasteur, Koch, Behring and Roux. The explanation for this doubtless lies in the lag which so often occurs between the discovery of scientific facts and their practical application. Only about 1904, the date of the Louisiana Purchase Exposition, do we find any advance in this direction, when the expectation of life of a white male child at birth had risen to 48.2 years. From this time on the tide has been rising steadily. At 1915, the date of the Panama-Pacific Exposition, the expectancy of life was 53 years; at 1933, the date of the Century of Progress Exposition, it was 60.5 years; at 1939 it is estimated that the span of life would be at least one year longer.

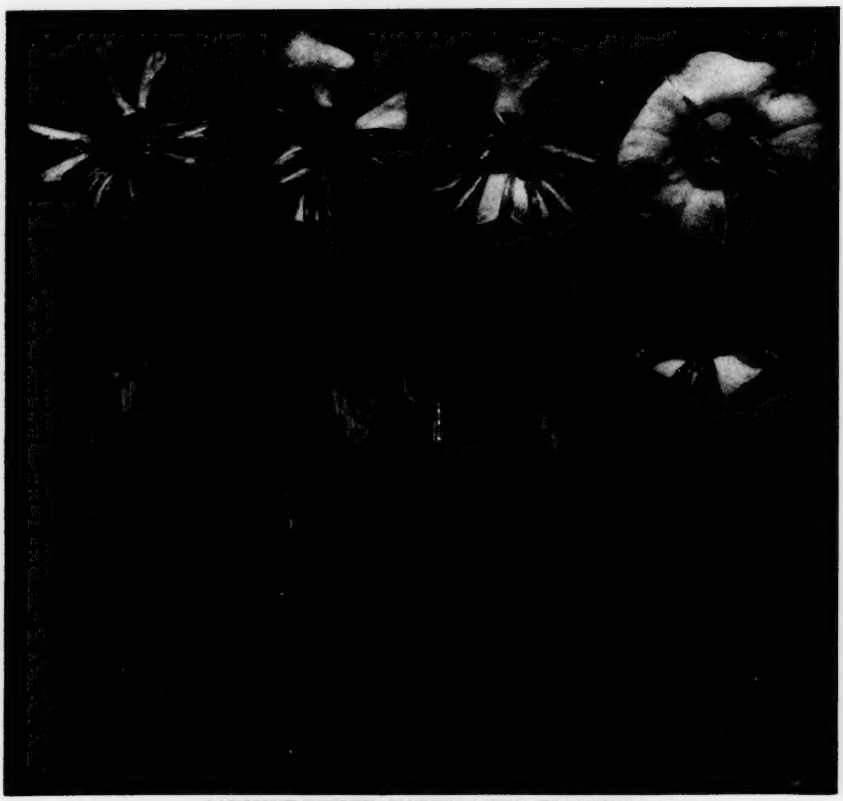
The prolongation of life among females has been even more striking. During the period between the Crystal Palace Exposition in New York in 1853 and the present New York World's Fair the expectation of life at birth among females in the United States has increased from 40.5 years to more than 65 years.

As has often been remarked before, the extension of the span of life has not been so spectacular in the case of young adults as among infants and children. The Metropolitan Company's survey finds that between 1853 and 1915 the expectation of life for both sexes, at the age of twenty-five, showed only small gains, though in the next two decades a definite improvement had taken place. — *J. Can. Med. Assoc.*, July, 1939.

INHERITANCE IN NICOTIANA TABACUM—XV

Carmine-White Variegation

K. R. STINO*



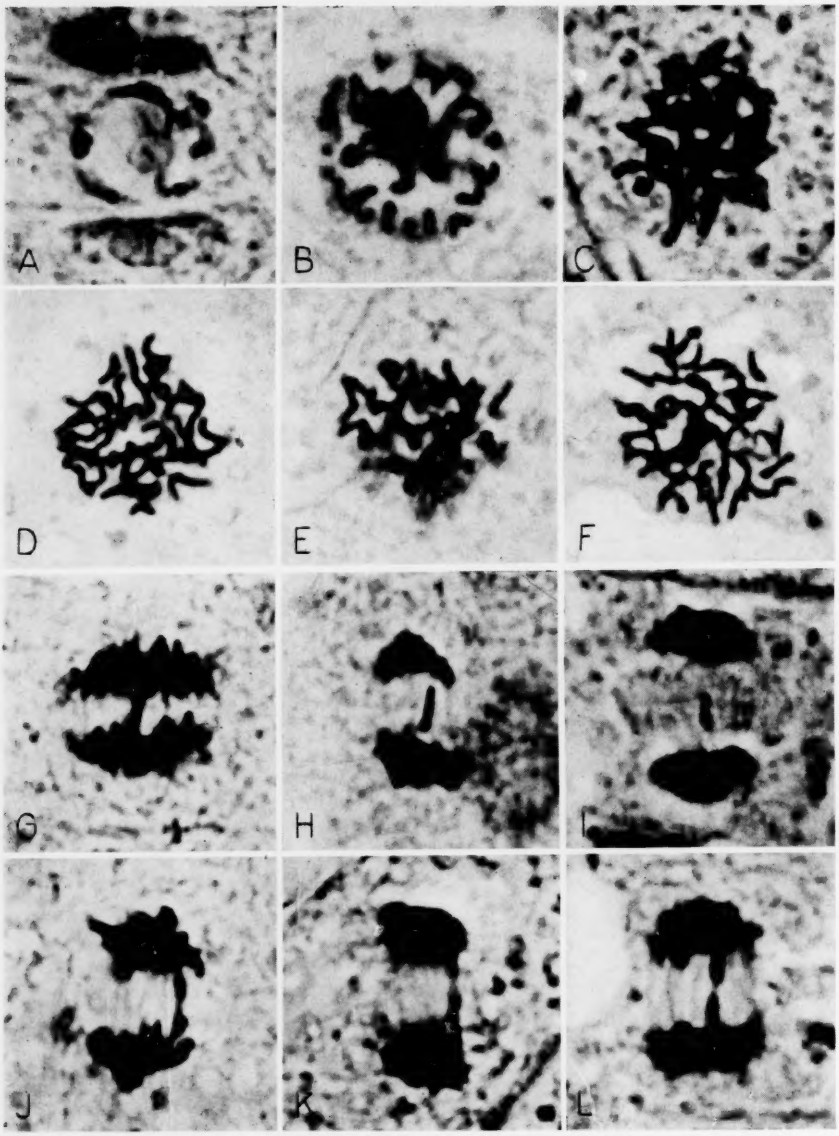
CARMINE-WHITE VARIEGATED FLOWERS

Figure 4

Photographs of flowers from the variegated plant showing varying amounts of white. The carmine sectors are flecked with white but the white sectors contain no flecks.

AMONG the monosomics of *Nicotiana tabacum*, haplo-*C* has been found rather frequently to produce new types having $23_{II} + C_I +$ a fragment. These fragments have been found to be translocation products between the *P* and *C* chromosomes, consisting essentially of one or the other arm of the *C* chromosome with a smaller or larger segment derived from the *P*

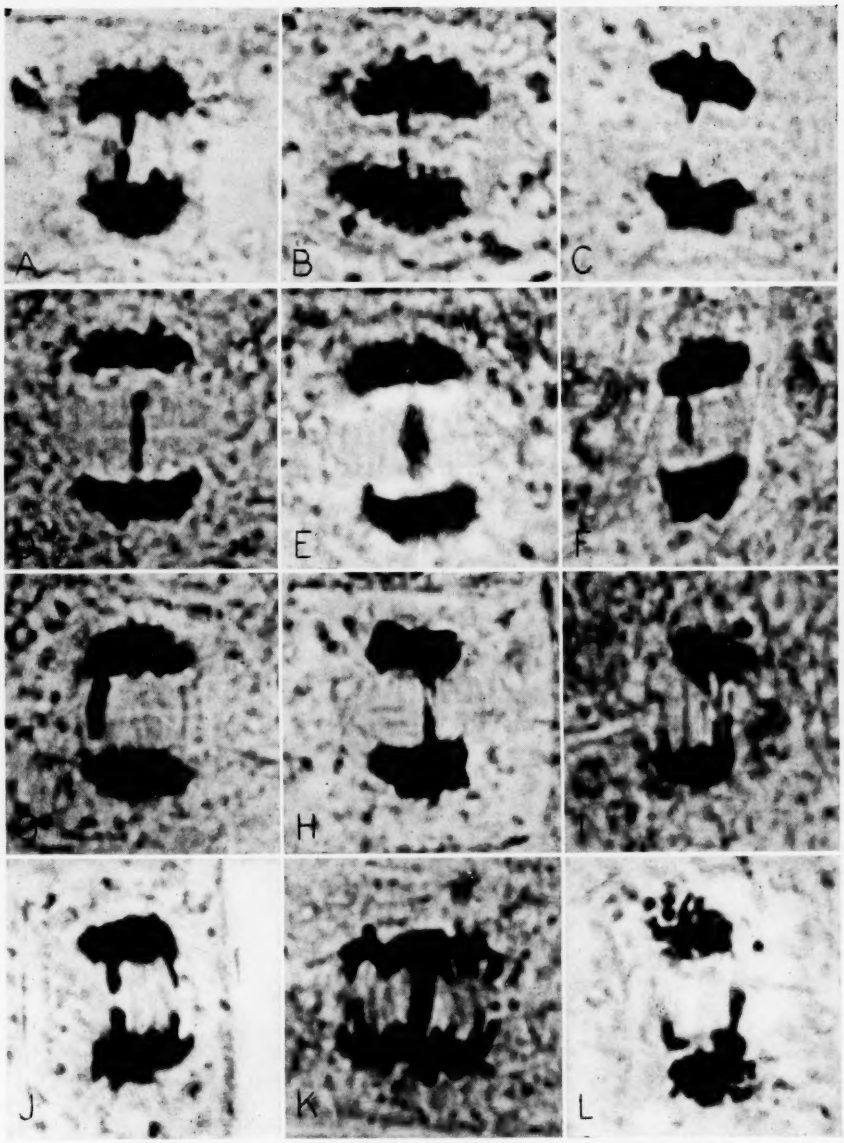
*This research was done in the Division of Genetics, University of California, Berkeley, under a Fellowship from the Egyptian Government. My sincerest thanks and gratitude are due Professor R. E. Clausen, who suggested and outlined the problem and supervised its execution.



RING STRUCTURE IN CHROMOSOMES OF CARMINE-WHITE VARIEGATION

Figure 5

Photomicrographs of individual cells from cross and longitudinal sections of root tips of the variegated plant (Figure 4). Magnification is about 2100 times. *A*—cross section showing the ring in prophase. *B*—cross section showing a larger ring in prophase. *C, D, E, F*—cross sections showing different sizes of the ring chromosomes at metaphase. *G*—longitudinal sections; early anaphase, double ring formation. *H*—longitudinal section; late anaphase, medium-size double ring lagging. *I*—longtitudinal section; early telophase, small double ring, lagging. *J*—longitudinal section; early telophase; a twisted, large double ring. *K*—longitudinal section; late telophase; twisted double ring. The cell plate will cut it unequally. *L*—late telophase; a twisted double ring severed at the mid-section.



COMPLEX RING STRUCTURE IN TOBACCO CHROMOSOMES

Figure 6

More photomicrographs of individual cells from root tips, showing double and interlocked rings. *A* is the same as Figure 5*L* except the lower part of the ring may be excluded from the nucleus. *B*—double ring cut into parts by the cell plate. *C*—disjunctional rings separated before plate formation. *D*—lagging, interlocked ring. *E*—lagging, figure 8 double ring. *F*—double ring which will be cut into equal parts by the cell plate. The lower section will be lost. *G*—same as *F* except that the cell plate will cut the ring into unequal parts. *H*—the double ring will be cut into equal segments and both will be included in the daughter nuclei. *I*—double ring already cut; the ends of the lower segment have not fused. *J*—two double rings already broken. *K*—two double rings lying close together. *L*—two rings interlocked. Both may be included in the lower nucleus.

chromosome attached to it.[6] Since the factor for flower color, *Wh*, is located in the *C* chromosome, fragment types may be identified as to which arm is present, by crosses with white, *wh*. In a cross between one of these fragment types of the constitution $23_{II} + (C\text{-}wh)_I + (fr\text{-}Wh)_I$ with normal white, $23_{II} + (C\text{-}wh)_{II}$, a single exceptional plant with carmine-white variegated flowers was found in the progeny. This plant is the subject of the present report.

The variegated plant was normal in size and vegetative characters. The variegation is the result of change from carmine to white in some sectors of the flowers (Figure 4), as is evidenced by the fact that the colored sectors are flecked with white, whereas the white segments contain no colored flecks. In this respect it differs from the classical instances of variegation such as those of *Antirrhinum*, *Zea*, etc., which exhibit colored flecks on a white background, indicating that the direction of change is from the recessive to the dominant condition. The expression varies widely in different flowers, some are almost carmine with white flecks, while others are entirely white, and at times branches bearing white flowers only may be produced. The condition is essentially the same as that of the carmine-coral variegation, which Clausen[1] ascribed to instability of a very small chromosome fragment bearing the dominant factor, rather than to any special condition or constitution of the gene. Miss McClintock[2] working with a similar type of variegation in *Zea*, demonstrated that ring chromosomes were present, and that, since they carried the dominant factor, their loss or unequal division was responsible for the phenomenon. Her later investigations[3] confirm this conclusion. The unstable chromosome fragment present in carmine-coral plants has also been found to be ring-shaped (Clausen, unpublished), although it is so small as to be difficult to study; and the present instance of carmine-white variegation is clearly associated with a ring-chromosome.

Observations

Meiotic studies of the variegated plant were made on aceto-carmine preparations of microsporocytes. At first metaphase $23_{II} + C_I + fr_I$ chromosomes were counted in 19 cells, while in six cells the fragment was not present. Although the fragment was usually unassociated, it was once seen conjugating with the *C* chromosome, but it is often difficult, due to its small size, to decide whether it is absent or conjugating. The size of the fragment varied in different cells; sometimes it was very small, at other times it was larger than the original fragment. It was impossible to demonstrate its ring shape in these preparations. At first anaphase the fragment was frequently found to lag, by actual count in 48 cells in 100. When the fragment lagged it remained undivided in most instances, but in a few it divided or seemed to break up into small fragments. The fact that it lagged in 48 per cent of the cells indicates that it was unassociated at least to that extent. If it conjugated with the *C* chromosome, chromatin bridges should appear at first anaphase, but none was observed. At second anaphase, however, chromatin bridges were sometimes seen; in other instances the fragment appeared to break into several pieces and other even more complex behavior was occasionally observed. In another slide prepared from a different bud the fragment was absent in all the 25 cells examined at first metaphase. This indicates that the fragment was lost at some early stage of development.

In order to study the behavior of the fragment in somatic divisions, cuttings made from side branches were treated for 24 hours with a commercial solution of heteroauxin and then planted in a mixture of peat and sand. Excellent root tips were collected two weeks later, fixed in chromoacetic formalin, dehydrated according to the Randolph[5] schedule and embedded. Longitudinal and cross sections were cut at 8, 10 and 12 microns and stained according to the Stockwell[7] technique. In cross sections the fragment was found to be ring-shaped. It

was easily seen in both prophase and metaphase (Figure 5, *A-F*). The size of the ring varied considerably in different cells. Rarely two rings were found in the same cell, while in many cells none was seen. In longitudinal sections over 100 cells in early and late telophase were examined; 42 per cent of them showed chromatin bridges. Different configurations of this bridge were readily detectable. Most of them were double rings (Figure 5, *G-L*); possibly some were interlocked (Figure 6, *D* and *L*) and some disjunctional (Figure 6*C*). Rarely two double rings were seen in the same cell (Figure 6, *J* and *K*). The rings were orientated medianly (Figure 6*H*), or non-medianly (Figure 6*G*), and accordingly upon formation of the cell plate they were cut into two equal or non-equal parts. In some cells a small double ring (Figure 5*I*) was seen lagging between the two daughter nuclei, leading to production of cells without rings. The broken ends of the double rings were often seen free and sometimes of unequal length (Figure 6*I*). If one portion of the double ring is not included in a nucleus (Figure 6, *F* and *G*), that nucleus will lack the ring, thus the carmine factor may be eliminated either by loss of the ring or possibly by its unequal division.

Extensive studies of transmission of the ring chromosome have not yet been made, but one progeny, 38300, of haplo-*C* white × carmine-white, consisted of 33 white and one carmine-white plant, indicating low transmission of the ring through the pollen. The single carmine-white plant seemed to be identical with the original variegated plant.

Conclusions

The behavior of this ring chromosome in somatic division agrees in most respects with that described by McClintock[3] in *Zea*. In both instances, double, interlocked and disjunctional rings were observed. Not only are smaller double rings lost by lagging but larger ones may also occasionally be eliminated (Figure 5*H* and 6*D*). Sometimes the broken ends of the double rings are seen clearly separated (Figure 6*I*), which suggests that if the chromosome is not split, fusion does not take place immediately after breakage. Otherwise, rod-shaped chromosomes should be produced from double rings. Such rod-shaped chromosomes have not been found in the cytological preparations, and the absence of self-carmine sectors in the variegated flowers also indicates that they are not produced. Since prophase and metaphase of root tip cells frequently exhibit rings, and at least forty-two per cent of anaphases in these same preparations exhibit double ring configurations, rings must evidently arise from fusion of broken ends. Variation in size of rings also supports this conclusion. However, the possibility that fusion of adjacent chromatids may occur, analogous to that demonstrated by McClintock[4] in meiotic cells, cannot be dismissed. Such behavior would give rod-shaped chromosomes with united ends, which would result in the same type of double ring configurations at anaphase as have been observed. The mode of division of ring chromosomes requires further investigation.

The carmine sectors in a given flower sometimes appeared to exhibit different color intensities. The darker portions may possibly consist of cells having more than one ring chromosome, and consequently more than one gene for the color factor; or having larger ring chromosomes with segments containing the factor duplicated. The observed range in size of the ring and the variation in number support this hypothesis.

Summary

Variegation in an exceptional plant of *N. tabacum* was found to be due to the presence of a large ring chromosome. In somatic cells, the ring varied in size and in number, while at anaphase, double, interlocked, and disjunctional rings were observed. In meiosis the ring looked like a fragment the size of which varied in different microsporocytes. Bridge formations were seen only in second anaphase.

(*Literature Cited on Page 24*)

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✦

MA-HIZ, MARISI OR MAIZE

The Greeks Had No Word for It

THE remarkable Inca and Maya civilizations were inseparably associated with Indian corn or maize, and this plant may be said to have been the basis of all pre-Columbian American agriculture. The importance of maize in present-day agriculture is known to all. Despite this importance, however, the origin of maize is shrouded in the mystery of American antiquity. It was evidently quite unknown to the Old World before the return of Columbus. In its present form, maize is entirely incapable of existing as a wild plant, and no clearly recognizable wild prototype has ever been authentically discovered. While the story of the origin of this cultivated plant has never been completely told, it is nevertheless an intensely fascinating one, and Mangelsdorf and Reeves have recently added a most significant chapter to it.* This well-written, thoroughly documented and penetrating analysis is noteworthy for its completeness. Evidence from Taxonomy, History, Paleobotany, Archaeology, Ethnology, Genetics, Cytology and other sources are summarized and applied to the problem.

Largely as a result of their own valuable studies of the cytology and genetics of hybrids involving various combinations of the three American genera, *Zea*, *Euchlaena* and *Tripsacum*, the authors are led to propose the rather novel hypothesis that *Euchlaena*, a wild genus, arose as a result of natural hybridization between cultivated *Zea* and some species of *Tripsacum*. It is suggested that cultivated maize has descended from a wild ancestor which was a kind of homozygous pod corn indigenous to the tropical lowlands of South America. This hypothetical prototype is now either extinct or unknown. Numerous arguments are adduced in favor of these hypotheses, but the reviewer finds them unconvincing as compared with those in favor of the alternative view that maize was developed under culture directly from wild *Euchlaena* (cf. this JOURNAL, 301:245-247, 1939). This, however, may be largely the result of a strong bias on the part of the reviewer in favor of simplicity in hypotheses: the reader must himself critically weigh the evidence.

Regardless of whether or not one agrees that the hypotheses about which the monograph centers have adequate support in fact, it is quite clear that this treatment is by far the most complete summary in print of the facts known about the interrelations of Indian Corn and its relatives. It will undoubtedly remain for a long time to come a standard source of reference for those interested in the origin and history of maize and the part played by this grass in American and World civilization. Even to those not primarily interested in the taxonomic, genetic and cytological details, the book can be recommended as decidedly "good reading." G. W. BEADLE

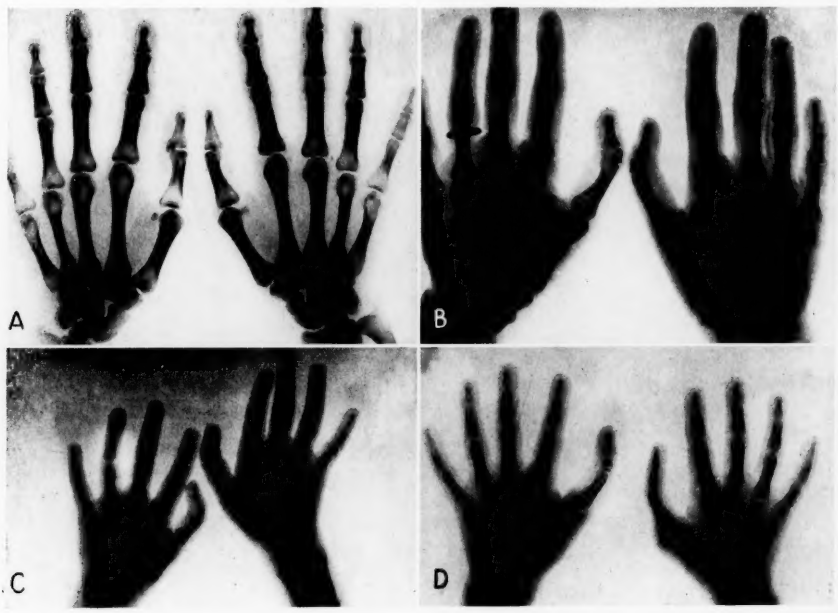
Stanford University

*MANGELSDORF, P. C., and R. G. REEVES, *The Origin of Indian Corn and Its Relatives* 315 p. Bull. 574 (Monograph), Texas Agric. Exp. Sta., 1939.

HEREDITARY POLYDACTYLY

Associated with Extra Phalanges in the Thumbs

R. A. HEFNER
Miami University, Oxford, Ohio



VARIATION OF DOMINANT POLYDACTYLOUS GENE
Figure 7

X-rays of the hands of four individuals in one pedigree, showing the variation in effect of the dominant autosomal gene for polydactyly and extra phalanges in the thumbs. *A* shows the hands of IV-7, on the pedigree chart, Figure 9, in which the deviation from normal is only in that the thumbs are long and pointed. *B* shows those of IV-5, in which extra phalanges are present in the thumbs. *C* (V-10) has hands like those of her mother while *D* (V-14) has had an extra thumb amputated from the region of the enlargement and the extra bone structure on the right hand. V-10 and 14 had extra toes also, shown in Figure 8.

INHERITED abnormalities of the thumbs have been studied by many observers and receive frequent mention in genetic and medical literature[1,2,3]. Manoiloff[4] describes a case of polydactyly of the hands and feet in which duplication of the thumbs and great toes accounts for the extra digits. The photographs and X-ray illustrations which accompany this report readily confirm this description. In addition the X-ray illustrations clearly show another abnormal condition which has not been emphasized by this Russian writer. The "thumbs," both regular and extra, are clearly not thumbs but fingers, each with three well defined phalanges, instead of the two phalanges found in the normal thumb. The only evidence that this condition was recognized by Manoiloff is found in the statement, "On the hands both thumbs must be considered as sup-

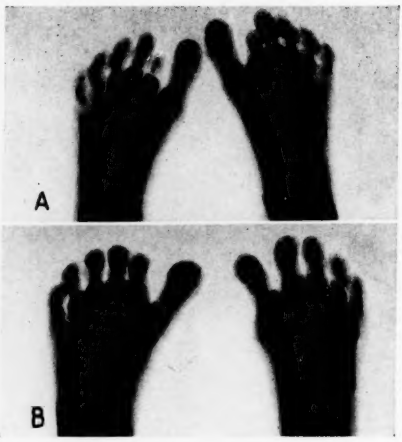
plementary fingers . . . They are connected not with one, but with two metacarpal bones."

The present report treats of a variable series of deviations from the normal anatomy of the thumbs, sometimes associated with polydactyly of the feet. Three types of deviations are observed:

I. The thumbs may be normal in number and equipped with two phalangeal joints. The only deviation is that the terminal joint is long and slender and tending to taper more or less to a point. Considered alone this condition would not be recognized as an abnormality, yet it seems to be inherited as a Mendelian dominant (Figure 7*A*).

II. The thumbs may be long and finger-like in appearance but fully apposable in the manner of a normal thumb. Thumbs of this type usually are bent at a very decided angle toward the index finger. The X-ray photograph of this bent thumb shows three phalanges, hence a "finger." (Figure 7*B*, *C*, and *D*.)

III. An extra thumb may be joined to the metacarpal of one or both of the thumbs described as type II (Figure 7*D*). This condition is accompanied by a supernumerary toe between the great toe and the normal second toe in all persons who show the extra thumbs in this family. These extra toes have three phalanges each. (Figures 8*A* and *B*.)



BOTH HANDS AND FEET ARE AFFECTED

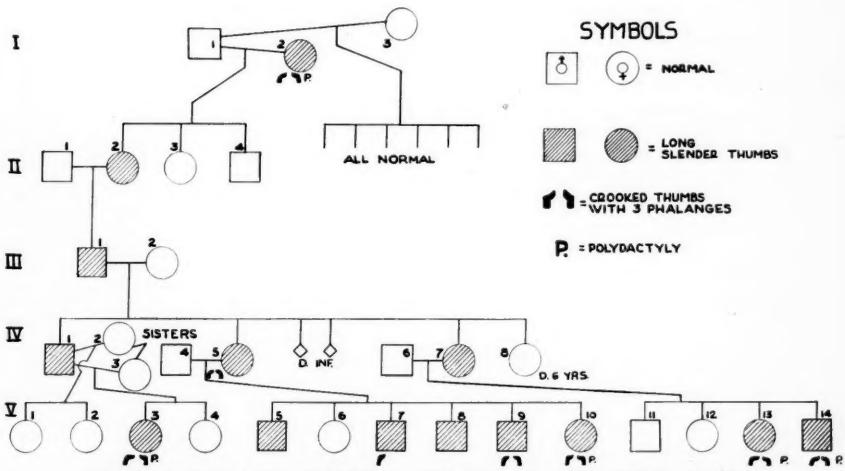
Figure 8

Extreme heterozygous expression of the gene for extra phalanges and digits was found in extra toes and fingers. These X-rays of the feet of V-10 (*A*) and V-14 (*B*) show the presence of extra toes, which have three joints and develop between the first and second normal toe. The normal large toe has been amputated from the right foot of V-14.

History of the Family

The family record of this series of conditions extends through five generations. The remote grandmother (Generation I-2) was said to have had long crooked thumbs, six fingers, and six toes. This obvious hearsay record was verified by two persons of advanced age now living. The hands of the grandson (III-1) of I-2 show the slender tapering thumbs of Type I. The thumbs of his mother were of like type. Two children (IV-1 and IV-7) also show thumbs of Type I. Another daughter (IV-5) has long slender thumbs bent at an angle toward the index fingers. An X-ray photograph (Figure 7*B*) shows three joints in these thumbs. The children of IV-5 show various types of normal and unusual thumbs as can be read from the chart (Figure 9). The person designated as IV-7 in the chart shows only the long pointed thumb condition (Figure 7*A*) as a slight deviation from normal. Two children of IV-7 show extreme deviations in the possession of six toes each and three phalanges in all thumbs. In addition, one of these children, V-14, had rudimentary extra thumbs at birth. A like condition occurred on one hand of a cousin, V-10 (Figure 7*C*) and on both hands of another cousin, V-3. These extra thumbs were joined to the metacarpal of the three jointed thumbs. All supernumerary thumbs were amputated at an early age.

In the most recent generation, it is remarkable to note that the polydactylous condition of the remote ancestor has reappeared in four children in three families. Three of these children were born within one year of each other. This coincidence is not explained by the application known hereditary mechanisms.



INHERITANCE OF ABNORMAL PHALANGES AND EXTRA DIGITS

Figure 9

Pedigree chart showing the dominant inheritance of a variable abnormality expressed by slender phalanges, extra phalanges, or extra fingers and toes. As is usually true in dominant abnormalities in humans, there have been no marriages of persons bearing the gene, so that only heterozygous individuals were observed.

The operation of recessive, multiple, or complementary factors would be inadequate since three separate, unrelated mates are involved in conjunction with three parents from the family concerned.

Summary

1. Manoiloff[4] illustrated three-jointed thumbs associated with Polydactyly.

2. The condition here described is that of the inheritance of a long slender thumb condition through five generations.

3. The inheritance seems to be that of a Mendelian dominant character.

4. Deviations from this thumb condition include polydactyly and thumbs showing three phalanges.

5. The recurrence of polydactyly in three families after an absence during three generations is not explained.

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THE SYMBOLIZING OF GENES AND OF CHROMOSOME ABERRATIONS

At the International Congress of Genetics held at Ithaca, N. Y., in 1932, it was resolved that the genetical societies of all countries should cooperate to prepare recommendations regarding the problem of standardizing genetical symbolism in order to discuss them at the next International Genetical Congress. The International Committee of Genetical Congresses appointed

Professor Dr. Tine Tammes (Groningen University) to take charge of the work to be done on behalf of this resolution; in cooperation with Dr. H. de Haan, Miss Tammes prepared a preliminary report on symbolism. Afterwards Miss Tammes has delegated the International Union of Biological Sciences to continue her task; in cooperation with the International Institute of Intellectual Cooperation at Paris, the Union convoked a meeting of delegates from various countries, which meeting was held in London in the hospitable home of the Linnean Society on August 14th and 15th, 1939. This meeting, under the presidency of Prof. Dr. M. J. Sirks (Groningen) was attended by the following delegates: Dr. A. Establier and Miss N. Nicolsky (from the I.I.I.C. at Paris), Prof. Dr. O. Winge (Denmark), Dr. B. Ephrussi (France), Prof. Dr. H. Nachtsheim (Germany), Prof. Dr. R. R. Gates, Prof. Dr. J. B. S. Haldane and Dr. A. E. Watkins (Great Britain), Prof. Dr. K. v. Körösy (Hungary), Dr. K. Ramiah and Dr. S. N. Venkatraman (India), Prof. Dr. M. J. Sirks and Dr. S. J. Wellensiek (Netherlands), Prof. Dr. O. L. Mohr (Norway), Miss Prof. Dr. M. Skalinska (Poland), Dr. O. Tedin (Sweden), Prof. Dr. F. Baltzer, Prof. Dr. E. Hadorn and Prof. Dr. A. Ernst (Switzerland), and Prof. Dr. E. W. Lindstrom (U. S. A.). The delegates from Belgium, Italy, Finland and Japan were unable to attend.

The following rules for symbolizing genes and chomosomes aberrations have been drawn up:

Choice of a Standard Type

If it is desired to establish a standard type, this should be the most common form, as a wild type, or if such cannot be determined, the first studied most dominant form, wild or cultivated.

SYMBOLS FOR GENES OF STANDARD TYPE. Generally +; for definite genes preferably the gene symbol with + as a superscript.

SYMBOLS FOR OTHER GENES. The smallest possible number of the initial letters of the name for the character for which latin is recommended when possible.

INDICATING DOMINANT AND RECESSIVE. Dominant capital initial letter; recessive small letter.

MULTIPLE ALLELES. The symbol of the first discovered allele, in small letters if recessive to standard type, with a capital initial letter if dominant to it. The standard type is designated by the same symbol with + as a superscript; the others by the same symbol with a special superscript in capitals for dominant, in small letters if recessive as compared to the first allele.

POLYMERIC (MULTIPLE) GENES. Genes which cannot be distinguished by the effects and for which the loci are not known, are symbolized by the same symbol with different Arabic figures as superscript A^1, A^2, A^3, or by the same symbol with the Roman figure of the chromosome as subscript A_I, A_{II}, A_{III}.

LETHAL GENES are indicated by the Greek letter Lambda (λ) which should be reserved for them, eventually as a subscript to another symbol, or separate combined with a Arabian figure with a Roman figure for the number of the chromosome, both as a subscript (λ_1, λ_2, λ_{IV}, etc.).

GENES IN POLYPLOIDS. When the chromosomes form polyvalents or if they pair at random, or if the segregation follows autopolyploid ratios, then the genes are written as many times as they are present, AAAa, AAaa, Aaaa, etc.

GROUPS OF LINKED GENES. The genes written in order from left to right in the chromosome and the symbols spaced II A b c D; between those of different chromosomes a semicoln (;).

GENES IN RELATED SPECIES. The same symbol but with a subscript of the abbreviation of the specific name.

FORMULAE. Generally AABbCc, but if parental gametic genotypes are known ABc/AbC and if a linkage group is concerned and the loci of the different genes are known $\frac{a\ b\ c}{d\ e}$, the female gametic genotypes being mentioned first.

RECIPROCAL CROSSES. In case plasmatic inheritance is involved an abbreviation of the name of the mother in parenthesis before the genotypic formula.

PRIORITY shall be valid if no essential objection to the symbol can be made.

LETTERTYPES. Italic for symbols of genes; roman letters for chromosome aberrations and rearrangements.

NOTE ON WOODS' PAPER ON PARENTAL INSTINCT

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Galton Laboratory, University College, London

IN a recent paper, Woods[2] studied parental instinct as revealed by records of European royalty contained in the *Almanach de Gotha*. In particular, an effort was made to discover evidence of a desire for male heirs, for it has been suggested that such a desire is the cause of the large families observed among royalty. The following quotation will reveal Woods' argument. "If this desire for a male offspring were exceedingly strong there would be many cases of daughters in the early years of marriage and then sons toward the latter years. To make this clear let us assume an extreme case. Suppose that as soon as one son appeared the parents . . . never had any more children. Then the distribution might be like this: Here are . . . ten fraternities, *A* to *J* . . .: *A-FFFM*; *B-FM*; *C-FM*; *D-FFFFFFM*; *E-FM*; *F-FFM*; *G-FM*; *H-FFM*; *I-FM*; *J-FM*.

"These results are . . . from the . . . throwing of a coin . . . (ignoring all one-male families) . . . Now suppose this tendency (or wish) is present though not in such an immoderate degree, then the same sort of distribution will make itself felt though to a lesser extent." In passing, it may be remarked that in providing his example, Woods suggests no reason for ". . . ignoring all one-male families," an extraordinarily arbitrary procedure.

Woods then listed a total of 46 sibships each of seven or more sibs, 17 from the first division of the *Almanach* and 29 from the second division, and partitioned these 46 sibships into those favoring the theory of the desire for male heirs and those against that theory. It is of interest to observe Woods' examination of these families. The first in his list is *MFMMMMFF*, and the following quotation shows Woods' approach in deciding whether this family is favorable or unfavorable to the theory. "The eldest is a son. This is followed by a daughter, then another son and then another. Thus by the fourth child there were already three sons and one daughter. By the fifth there was another son. Surely there was then no need to continue offspring in order to secure male succession. Again another son was born and he was followed by two more who were girls. Such cases as this are obviously against a theory that the large families are in part due to desire for males." It is to be noted that Woods thought four sons certainly enough to secure the succession, but it may well be that royal parents hold the less arbitrary view that more is better. At least, the only way to have many sons is to have many children. In any case it is curious that Woods regarded such families as *FFFMMFMFMM* and *MMFFFMMMM* as favorable to the theory, showing as they do a continuance of births after the birth of the fourth male. Also it is difficult to see, remembering the two families just mentioned, why *MFMMMFMM* and *MMFFMMM* or *FMMFMMMM* should be regarded as contrary to the theory. Further, a family *MMFFFFF* would seem to be one in which the parents wanted more males, but Woods judged it contrary to the theory. These examples show that Woods' partition is arbitrary and subjective. Since the data are themselves quite objective, a wholly objective examination is possible and Woods' partition should be rejected. In passing, it may be noted that Woods' partition of the 29 families of the second division shows 16 favoring and 13 against the theory, but in the text Woods made it 18 and 11, and in a footnote it appears as (17 and) 12. Further, the 17 fami-

lies of the first division were regarded as seven favoring and ten against, so that the correct totals seem to be 23 favoring and 23 against among 46, rather than 28 and 28 among 56, as stated by Woods. From these results, Woods concluded that there was little or no evidence that the large families of royalty result from a desire for male heirs.

Having concluded that large royal families do not result from the desire for male heirs, Woods suggested that ". . . it still may be that the small ones are.", that is, that the smaller families may result from family limitation following the birth of one or two sons. From his examination of the data, Woods concluded that this is in fact so, basing his purported proof on two features of the data. One is the high sex ratio observed, and the other is the tendency for the youngest member of a sibship to be male. I quote again from Woods, to make clear his argument. "When males are in sufficient number . . . birth-limitation does often take place as an analysis of the sex ratios and sequences in the small families virtually proves. First, a list of the fraternities of four . . . revealed that a total of 116 births gave 65 males to 51 females. Furthermore if the last of four children tends to be a son it is corroborative evidence — the idea being that the heir now surely has arrived and therefore no more children. Of course, all this is bound up with complicated personal and family problems, matters of which we necessarily know nothing, but the statistical test rises out of it all to demonstrate something which while not true in each instance is true in the average. There are twenty cases where the ultimate is a boy and only nine where a girl is the last child." It is regrettable that there is no further mention of the statistical test.

The first feature of the data advocated by Woods as proof of a preference for males is the excess of males over females. Unfortunately this excess cannot be regarded as such a proof, for it is not a theoretical consequence of the preference suggested. As would be admitted by all but the superstitious, a desire for males will not increase their relative frequency. However, it might be thought that a tendency for reproductive efforts to cease as soon as the family has the desired number of boys would result in an excess of males over females. This is wholly fallacious, as may easily be seen from just such an example as that given by Woods in which each family consisted of an unbroken sequence of females followed by a single male. Woods' example is from coin tossing but it could equally well be from any distribution of two alternatives not necessarily of equal theoretical frequency. Thus if a series of alternatives be recorded, they will occur in approximately their theoretical frequencies, and these frequencies cannot be altered by dividing the series into "families," whatever kind of family may be employed. Of course it is necessary

TABLE I. Sex ratio for youngest children and for their sibs.

	♂	♀	Total
Last children	99	63	162
Earlier children	145	112	257
Total	244	175	419

TABLE II. Fertility in small royal families.

	♂	♀
Sovereign Houses		
Families of four	65	51
Families of three	64	47
Families of two	56	30
Families of one	13	8
Mediatized Princes		
Families of two	59	47
Families of one	21	20
Total	278	203
Last children	99	63
	377	266

	♂	♀
Last child	20	9
Last child	20	17
Last child	28	15
Last child	31	22

to retain families of one child each as well as those of other sizes.

The second feature of the data advocated by Woods as proving a preference for males is the tendency for the youngest member of a sibship to be male, as if the parents went on until they had as many males as they desired or until other factors supervened. If such were the nature of the data, it might reasonably be maintained that the parents had continued until they had the desired number of males, but it must be noted that such data are equally convincing as proof of the hypothesis that parents like the youngest child to be male. If it were true that parents continued until they had the desired number of males, there would be a tendency for the last child to be male. However, such a practice would tend not merely to make males more frequent than females as youngest children, but to make the frequency of males as youngest children exceed that to be expected by random sampling. Thus, taking the families of two, three or four children, a 2×2 table may be constructed, as Table I, showing the numbers of the two sexes separately for last children and for all earlier children. For Table I, the corrected chi-square is 0.716 with one degree of freedom. Since a value as large would be encountered about two times in five in random sampling from a population in which the sex ratio is 244:175 among last children and among earlier children, the sex-ratio among last children cannot be regarded as differing significantly from that of earlier children, although it may be observed that the deviation is in the direction to be expected if there is a tendency for youngest children to be male.

To conclude this paper, Woods' Table II regarding "small" families is reproduced here as Table II, and the data of the table are examined. Before proceeding to examine the data of Table II, a serious statistical error in Woods' summary of the data should be noted. The summary figures 377 and 266 are misleading, the totals for last children being included twice.

The first step in examining Table II is the formation and examination of Tables III and IV. Table III is a $2\times2\times2$ table showing the distribution of children by sexes, by divisions of the *Almanach*, and whether they were last children or not, for families of two. Table IV is a 2×2 table comparing sex-ratio between sovereign houses and mediatized princes in families of one.

The examination of Table III follows the procedure set forth by Bartlett,[1] and finds a chi-square of 0.00714 with one degree of freedom, corresponding to a probability of 0.93. For Table IV, chi-square is 0.282 with one degree of freedom, corresponding to a probability of 0.60. Tables III and IV are therefore homogeneous and the distinction between the two divisions of the *Almanach* may be dropped without loss of information. It is now possible to form Table V which shows the distribution of sexes by family size for youngest children and for their sibs.

The examination of Table V also follows Bartlett, and chi-square is found to be 2.478 with two degrees of freedom, corresponding to a probability of 0.29. Table V is therefore homogeneous and may be reduced by addition in any and

TABLE III. Sex ratio in families of two, for youngest children and for their sibs, by divisions of the *Almanach*.

	Sovereign houses		Mediatized princes	
	♂	♀	♂	♀
Earlier children	28	15	28	25
Last children	28	15	31	22

TABLE IV. Sex ratio in families of one, by divisions of the *Almanach*.

	♂	♀	Total
Sovereign houses	13	8	21
Mediatized princes	21	20	41
Total	34	28	62

TABLE V. Sex ratio by family size, for youngest children and for their sibs.

Family size	Earlier children		Last children	
	♂	♀	♂	♀
4	45	42	20	9
3	44	30	20	17
2	56	40	59	37

each of the three possible ways. Ignoring family size, Table VI of this paper is formed, and it has already been discussed. There is no interest in ignoring sex, for it is obvious that the last child represents a smaller proportion as family size increases. However, we may ignore birth order and inquire whether sex-ratio is the same for families of different size. In this case it is possible to bring in families of one, the data appearing as Table VI.

For Table VI, chi-square is 0.718 with three degrees of freedom, corresponding to a probability of 0.84. However, it might be asked whether there is a regression of sex-ratio on family size, and to answer this question it is necessary to examine the single degree of freedom corresponding to that regression. We are spared the effort of performing this test, for the one degree of freedom cannot have a chi-square exceeding that found for all the three degrees of freedom of the whole table, and since a chi-square of 0.718 is too small to be significant for one degree of freedom, there cannot be a significant regression of sex ratio on family size in the data of Table VI.

There remains one point of interest—the sex ratio in the whole of the data, 278 males to 203 females. These data obviously depart from the hypothesis of equal frequency of the sexes, but since it is known that the sexes are not equally frequent, the simplest procedure will be to give fiducial limits for the sex ratio. It is well-known that in a simple dichotomy of n individuals, expecting np of one kind of nq of the other, observing a and b respectively, where $a + b = n$, and for $a > np$, the corrected chi-square is given by

$$\chi^2 = \frac{(a - np - \frac{1}{2})^2}{npq}$$

This equation may be solved for p, finding

$$p = \frac{\alpha + \chi^2 \pm \sqrt{\chi^2 (\chi^2 + 2\alpha - \frac{2}{n})}}{2(n + \chi^2)}$$

where for convenience $2a$-1 has been replaced by α. The lower limit on p corresponding to a fiducial probability π is then

$$\overset{\vee}{p} = \frac{\alpha + \chi_\pi^2 - \sqrt{\chi_\pi^2 (\chi_\pi^2 + 2\alpha - \frac{2}{n})}}{2(n + \chi_\pi^2)}$$

In the data above, $a = 273$, $b = 203$, so that the one per cent lower fiducial limit of p, the probability of birth of a male, is 0.51824, corresponding to a sex ratio of 107.6:100. Thus there can be little doubt that the families included in these data belong to a population having a sex ratio substantially higher than that of mankind generally, which is usually observed to be about 105:100.

TABLE VI. Sex ratio by family size.

Family size	♂	♀	Total
4	65	51	116
3	64	47	111
2	115	77	192
1	34	28	62
Total	278	203	481

Conclusions

It has been shown that Woods' study of data regarding royal families is arbitrary and subjective in the case of "large" families, and fallacious in the case of "small" families. His data for small families are examined and it is found that there is no association between family size, sex, birth order, and rank by divisions of the *Almanach*, and that the data show no tendency for youngest children to be male. Finally, it is shown that the observed sex ratio of 137:100 exceeds the value of 105:100, usually observed, at the one per cent fiducial level.

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FERTILITY AND CHROMOSOME LENGTH

Correlations Between Chromosome Length and Viability of Gametes in Autopolyploid Plants

D. KOSTOFF

IN my previous publication[8] I advanced the theory that autopolyploids obtained from plants with long chromosomes should have lower fertility than those obtained from plants with short chromosomes, provided that in either case the plants have the same or approximately the same chromosome numbers. The theory is based upon the chiasma frequency in long and short chromosomes.

The investigations upon the chiasma frequency in long and short chromosomes[4,5,6,10,12,13] and even in long and short arms[1] of one and the same chromosome, showed that the mean number of chiasmata per bivalent and per arm is approximately proportional to the length of the pairing blocks. This ratio breaks down for very short chromosomes,[2,3,11] i.e. when there is a wide range in chromosome length, but even in these cases, long chromosomes have more chiasmata than the shortest one. Long chromosomes in autopolyploid plants form quadrivalents more frequently than the short ones.[5,7,9,14] Very short chromosomes usually form bivalents. Polyvalency (quadrivalency and trivalency with univalency) leads as a rule to abnormal chromosome distribution during meiosis, this being an indirect cause for the reduction in the viability of the gametes and further for the reduction of the fertility of "autotetraploids" and "autooctoploids."

The data given in the table support the theoretical deductions. Autotetraploid plants having short chromosomes (*Beta* and *Taraxacum*) form the largest percentage of viable pollen, the autotetraploid plant *Festuca pratensis* having long chromosomes forms the largest percentage of abortive pollen, while *Nicotiana* autotetraploids having medium chromosomes form much more viable pollen than *Festuca* and less viable than *Beta* and *Taraxacum*.

TABLE I. Viability of the pollen grains in diploid and tetraploid plants with various chromosome lengths.

Plants	Somatic chromosome numbers	Per cent abortive pollen
A. PLANTS WITH SHORT CHROMOSOMES		
1. *Beta vulgaris* diploid	18	3.4%
2. *Beta vulgaris* tetraploid	36	4.1%
3. *Taraxacum kok* — Saghys diploid	16	1.3%
4. *Taraxacum kok* — Saghys tetraploid	32	4.1%
B. PLANTS WITH MEDIUM CHROMOSOMES		
1. *Nicotiana alata* diploid	18	8.6%
2. *Nicotiana alata* tetraploid	36	23.1%
3. *Nicotiana trigonophylla* diploid	24	11.4%
4. *Nicotiana trigonophylla* tetraploid	48	23.5%
C. PLANTS WITH LONG CHROMOSOMES		
1. *Festuca pratensis* diploid	14	7.8%
2. *Festuca pratensis* tetraploid	28	46.6%

Viability of gametes is more affected in tetraploid plants with large chromosome numbers than in plants with small chromosome numbers. *Nicotiana rustica* ($2n=48$), for example, has medium chromosomes. Tetraploid *N. rustica* with 96 somatic chromosomes has a rather high percentage of abortive pollen (56.6%) and its fertility is greatly reduced. *Triticum vulgare* ($2n=42$) and *T durum* ($2n=28$) have long chromosomes. Autotetraploid *T. vulgare* and autooctoploid *T. durum* are self sterile as a consequence of reduced gamete viability.

It seems quite reasonable to conclude that from an evolutionary point of view polyploid plants with large chromosome numbers have originated from plants with relatively short chromosomes and that those having long chromosomes and large chromosome numbers usually are allopolyploids.

Autotetraploids produced from plants with short chromosomes and with small chromosome numbers will also be more suitable for plant breeding work.

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UP THE LADDER

SEVERAL years ago Dr. Siegel wrote a book which was reviewed rather critically in this Journal. That book bristled with unfortunate errors, misstatements and misinterpretations about eugenics which were both harmful and misleading, in spite of a ringing foreword by a university Chancellor which touted the book as truly Pentecostal. Now Dr. Siegel has done another book.* Oddly enough this improved effort got neither a foreword nor a publisher. It is a pleasure to report that it is much better than his first effort, but is still not perfect by any means. Errors still abound, but they are not as serious as those in the previous volume. Some of the typographical errors are real contributions to the muddled battle of words which goes on constantly around and about *Homo Sapiens*, and which can be appreciated by one who within the year gave the world a page-full of "robbins." Thus in discussing race (P. 63): "The excessive strain (on the 'selected intellectuals') has made them somewhat exhaustive and as a result they become less fertile, while the inferior group having very few responsibilities retained their strength and virility and had larger families." Much of the sound and fury on race at the moment leaves this reviewer definitely *exhaustive*, too! — R. C.

*Siegel, M. *Population, Race and Eugenics.* Pp. 206. Published by the author. 1939.

ROOT-KNOT RESISTANCE IN BEANS

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THE root-knot disease caused by the nematode *Heterodera marioni* (Cornu) Goodey is of widespread occurrence on a vast number of species in the tropics and warmer portions of the temperature zones including southern United States. Among the more susceptible hosts is the common bean, *Phaseolus vulgaris* L. Isbell[4] described two strains of this species which he found to be highly resistant to the formation of root-knot galls in tests at Auburn, Alabama. In 1938 one of these strains was introduced by the Alabama Agricultural Experiment Station under the name of Alabama No. 1, and it is now available from commercial seedsmen.

In this paper the term "root-knot resistant" is used in describing Alabama No. 1 in place of the term "nematode-resistant" as used by Isbell.[4] It has been found[2] that this variety and a number of other plants which produce few or no root-knot galls when grown in nematode infested soil are fully as subject to the entrance of nematode larvae into their roots as root-knot-susceptible hosts.

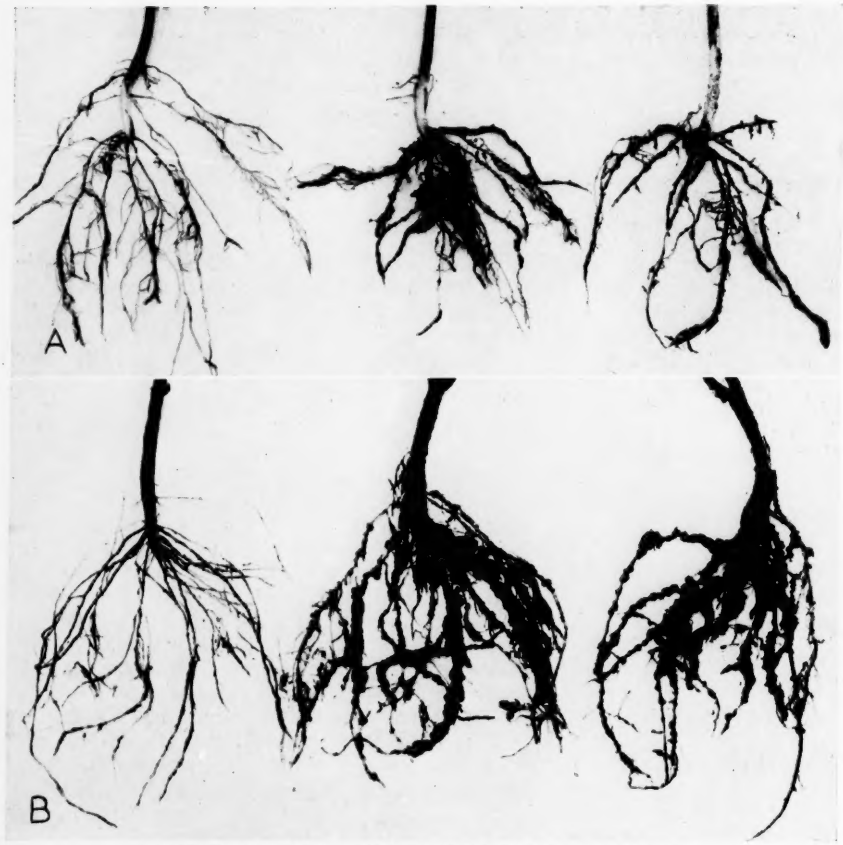
Bean breeding experiments designed to combine the root-knot resistance of Alabama No. 1 with desirable traits possessed by other pole bean varieties have been undertaken by the writer. A genetic study has also been conducted on the inheritance of root-knot resistance in a cross between Alabama No. 1 and Kentucky Wonder, a very susceptible variety.

Alabama No. 1 has heavy anthocyanin pigmentation in the stems and leaves of all stages. This character is dominant to the non-pigmented condition in Kentucky Wonder. The latter was used as the female parent in most crosses, so the true F_1's could be distinguished from accidental selfs on the basis of their inheritance of anthocyanin pigmentation from the dominant male parent. One reciprocal cross was also completed.

Field soil was inoculated with the root-knot nematode prior to planting in a manner already recorded by the writer.[3] Greenhouse facilities were used only for seedling tests which were conducted according to a method previously described.[1]

Three seeds each of four different crosses of Kentucky Wonder ♀ × Alabama No. 1 ♂, and also two seeds of one reciprocal cross, Alabama No. 1 ♀ × Kentucky Wonder ♂, were sown in artificially inoculated soil in a greenhouse bench. A number of seeds of each parent variety were planted in the same bench for purposes of comparison. Twenty-two days after sowing, the roots were dug, washed free of soil, and examined for infection. All F_1 generation plants appeared susceptible to root-knot. There was little, if any, difference in the degree of root-knot infection observed on the susceptible parent and the F_1 hybrids. The validity of the crosses was established by the fact that the Kentucky Wonder ♀ × Alabama No. 1 ♂ hybrids possessed pigmentation similar to the male parent, and the hybrid of Alabama No. 1 ♀ × Kentucky Wonder ♂ was as susceptible to root-knot as the male Kentucky Wonder parent. A typical root of an F_1 plant and one of each parent taken from the seedling test is shown in Figure 10*A*.

As differences in apparent resistance to root-knot of seedlings and adult plants have been noted in the lima bean (*Phaseolus lunatus* var. *macrocarpus* Benth.)[1] it was considered advisable to grow further F_1 plants to maturity in inoculated soil under field conditions. Two seeds of one of the crosses and three of another were available for the adult-plant test. The roots were dug after the plants had produced a quantity of mature seed. All five were found to be susceptible to root-knot, and comparisons



RESISTANCE AND SUSCEPTIBILITY TO ROOT-KNOT

Figure 10

Roots of young seedlings (*A*) and of adult plants (*B*) of Alabama No. 1, the variety resistant to root-knot (at left), a susceptible variety, Kentucky Wonder (at right), and of the hybrid between these two varieties (center). Note that the hybrid is as heavily affected as the susceptible parent, showing the recessive inheritance of resistance. Segregation in the second generation gave some susceptible and some resistant plants.

with Kentucky Wonder plants grown under the same conditions indicated that there was no pronounced difference between the degree of infection on the F_1 and the susceptible parent. The Kentucky Wonder plants had galls that were slightly larger than those on the F_1, but for all practical purposes the latter was considered susceptible. Figure 10*B* shows a typical root of the F_1 and the two parents grown to maturity in infested field soil. These experiments involving reciprocal crosses clearly indicate that resistance to root-knot as possessed by Alabama No. 1 bean is inherited as a recessive trait.

A seedling test was made of an F_2 population involving more than 1,000 plants; however, a reliable classification of the plants on the basis of their resis-

tance and susceptibility to root-knot could not be obtained because of the severity of a stem-rot disease in this particular test. A large proportion of the plants appeared susceptible, indicating a genetic difference of more than one factor. An additional F_2 population of 208 plants all from a single cross was grown in artificially inoculated soil in the field. Examination of a planting of Kentucky Wonder and other susceptible varieties grown in adjacent rows, indicated that a fairly uniform infestation of the root-knot nematode was obtained in this test. Apparent variations in the degree of susceptibility were noted in this F_2 population, and the following classification was made: susceptible 144; intermediate 47; resistant 17.

In order to check the reliability of this classification F_3 progeny of the 47 intermediate and 17 resistant F_2 plants were grown in a greenhouse seedling test. Twenty days after planting the lines were classified as resistant, segregating, or susceptible. Variable lines that showed less than ten per cent of resistant plants were classified as susceptible. It was found that 12 of the 17 progeny of plants classified as resistant in the F_2 generation were true breeding for resistance in the F_3 seedling test, while four of the remaining five lines were classified as segregating and one as susceptible. Of the 47 F_3 lines from plants which were intermediate in the F_2 generation 36 were classified as segregating and eleven as susceptible. Thus after adjusting the F_2 data on the basis of the F_3 progeny tests a segregation of 156 susceptible; 40 intermediate; and 12 resistant was obtained.

This segregation constitutes almost a perfect fit to a 12:3:1 ratio as indicated by the extremely low χ^2 value of .103. However, such a hypothesis, which assumes the presence of two recessive genes for the production of root-knot resistance, is unsatisfactory because it would necessitate the occurrence of homozygous intermediate individuals possessing one pair of genes in the dominant and one pair in the recessive state. Out of the 47 progeny lines from plants classified as intermediate in the F_2 generation all of those not found to be susceptible were segregating further into susceptible, intermediate, and resistant classes.

It appears more reasonable to postulate an 11:4:1 ratio which when compared with the observed segregation gives a χ^2 value of 4.027. As five per cent significance in this test is 5.991 it may be considered that the observed data fits an 11:4:1 ratio reasonably well. Such a hypothesis assumes that two recessive genes are involved in the production of root-knot resistance and that they act in a quantitative manner with only those plants possessing one dominant gene having the intermediate phenotype. This hypothesis is suported by the fact that the heterozygous F_1 generation was practically as susceptible as the susceptible parent.

Using the symbols *R* and *R′* to designate the two genes one would have the following genotypic ratio in the F_2 generation according to the 11:4:1 hypothesis:

11	SUSCEPTIBLE		
	1 RR R′R′	4	Rr R′r′
	2 Rr R′R′	1	RR r′r′
	2 RR R′r′	1	rr R′R′
4	INTERMEDIATE		
	2 Rr r′r′	2	rr R′r′
1	RESISTANT		
	1 rr r′r′		

According to this hypothesis one would expect intermediate F_2 plants to segregate in a ratio of 1 susceptible: 2 intermediate: 1 resistant in the F_3 generation. Accurate classification into the susceptible and intermediate phenotypes was difficult in the seedling tests; however, approximately 30 per cent of all plants from lines classified as segregating were classed as resistant. This deviation of only five per cent from the theoretical expectation may be accounted for by the probable occurrence of plants which escaped infection in the seedling tests.

Of the susceptible genotypes in the F_2 generation only those which, like the F_1, were heterozygous for both genes would show a phenotypic segregation in the F_3. This segregation should approximate an

11:4:1 ratio. An accurate check of this was not possible because of the difficulty of distinguishing susceptible from intermediate plants. However, a later seedling test, involving a large number of F_3 progeny of additional F_2 plants, which were not tested for resistance, indicated that about one-third of the lines not classified as resistant or undergoing a monohybrid segregation were showing considerable variation with a few plants appearing resistant.

This type of inheritance of root-knot resistance is in reverse of that found in cowpeas, *Vigna sinensis* (Torner) Savi, by Orton.[5] In a cross between the resistant variety Iron and certain susceptible varieties he found resistance to be inherited as a dominant trait with an F_2 segregation that was too complicated for explanation on a monohybrid basis.

Summary

It may be concluded on the basis of these studies that resistance to root-knot in Alabama No. 1 bean is inherited as a double recessive trait. It also appears probable that the inheritance is on a quantitative basis with all individuals possessing two or more dominant genes appearing susceptible to root-knot, and those with one dominant gene appearing intermediate. An F_2 segregation of approximately 11 susceptible: 4 intermediate: 1 resistant was obtained.

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✦

DANZIG HONORS SCIENTISTS

A STAMP IN HONOR OF GREGOR MENDEL

Figure 11

A bit of genetic philately deserves to get into the record before it is entirely swallowed up in the turmoil of World War II. During the spring of 1939, while hopeful souls were building a new bridge across the Polish Corridor, "the Free City" of Danzig released a series of large size stamps honoring scientists who had contributed to the advance of biology and to human welfare. The ten pfennig stamp of this series which is reproduced here is a reproduction of perhaps the most natural portrait of Gregor Mendel. The stamp is an excellent reproduction, in a red-brown ink. The inscription, translated "healthy children," and "a happy future," has been given perhaps a tragically sardonic implication when we consider how grimly modern warfare deals with both our biological and our economic future. The other two stamps in the series issued before Danzig was reunited to the Fatherland commemorated Koch and Röntgen. This appears to be the first time that the founder of genetics has been honored by the postal officials of any country.

THE PRODUCTION OF POLYPLOIDS IN GOSSYPIUM

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THE interest in producing polyploids in Gossypium was stimulated by the usual advantages cited for polyploids: greater size, vigor, hardiness, etc.; and, in addition, as polyploidy usually increases cell size, it would be expected to change profoundly the length and diameter of cotton fibers, factors closely related to their utility.

Following the work of Randolph,[10] Dorsey,[4] and Sax[12] one series of cotton ovaries was treated with hot water, another with ice water, and others in boxes with controlled high and low temperature. The chromosome number in seedlings from the treated ovaries was determined from aceto-carmine smears of root tips. Only one polyploid was found in over 600 seedlings, and it was in a 13-chromosome Asiatic species, *G. herbaceum* L. This autotetraploid, found in March, 1937, came from a capsule treated one hour in ice water, 90 hours after pollination. The plant grew, and grafts are still living. Several sterile hybrid plants were treated 10 to 15 times in ice water and in cold and hot chambers in an attempt to induce fertile polyploid branches, but no fertile flowers appeared.

In the summer of 1938 a number of hybrid Gossypium seedlings and grafts of hybrids were treated with colchicine, after Blakeslee and Avery[2] had shown that this alkaloid produces a high percentage of polyploids in Datura. Some shoots that grew from the treated meristems showed evidence of having double the normal number of chromosomes, the evidence being darker green leaves, larger hairs on leaves, and more stubby shaped flower buds. The stomata of leaves from these branches were larger than stomata of leaves on untreated plants. The branches that showed evidence of polyploidy proved to have approximately double the normal number of chromosomes.

To treat cotton plants with colchicine a small slit is made about two cm. below the growing tip, and the tip is immersed in a vial containing 0.2 per cent aqueous solution of colchicine. With a temperature of 70 to 80 degrees shoots survive a 24 hour treatment, but a similar treatment at higher temperatures is lethal to some meristems. Seedlings are treated about the time the first internode begins to elongate. It is well to let cotyledonary buds grow, since many of them have their chromosome number doubled. After meristems of larger shoots were treated, it was occasionally observed that buds arising below the treated parts gave rise to branches with double the normal number of chromosomes, in some instances even after the tips were killed. It is necessary, however, to remove buds that arise three or four cm. below the treated tip, because untreated buds usually outgrow treated buds.

The chromosome number was doubled in branches of 55 per cent of the plants from seedlings of F_1 *G. hirsutum* L. (American upland) $\times$ *G. Barbadense* L. (Pima) that survived a 24-hour treat-

*This work was initiated and the part on the production of polyploids with high and low temperatures was done at Harvard University under the direction of Professor E. M. East. The author is grateful to Professor Karl Sax of the Arnold Arboretum, Harvard University, for the use of his controlled temperature chambers, and to Dr. Thomas Kerr for his help in preparing the manuscript. The author was agent for the Bureau of Plant Industry, U. S. Department of Agriculture and the North Carolina Experiment Station at the time the work with colchicine was done and the paper written.

ment in 0.2 per cent aqueous solution of colchicine. As both of these cottons are allotetraploids with 26 pairs of chromosomes, the induced polyploid is an octoploid with 52 pairs of chromosomes. Apical meristems of 25 grafts of triploids from American Upland × Asiatic, *G. hirsutum* × *G. herbaceum*, were treated with colchicine and eleven of them produced fertile flowers. This doubling of chromosome numbers in triploids of American 26-chromosome × Asiatic 13-chromosome gave hexaploids with 39 pairs of chromosomes. Exact data are unavailable on the percentage of chromosome doubling induced in other types that were treated, but it was above ten per cent in every case.

To date the chromosome number has been doubled with colchicine in eleven types of Gossypium to give tetraploids, hexaploids, and octoploids (Table I). From these polyploids, and the one produced by cold treatment, hybrids were made to give other polyploids including pentaploids, and heptaploids (Table II), which, with hybrids between species, give a complete series in Gossypium from diploid to octoploid.

Formulas for Describing Chromosome Composition of Polyploids

It is necessary to devise some way of describing the induced polyploid types of Gossypium. The limitations of the terms allo- and auto- polyploid and haploid are apparent when an attempt is made to describe and show the relationships between (1) several distantly related diploid species, (2) several natural allotetraploid species, (3) allotetraploids and hexaploids produced by doubling the chromosome number in species hybrids, (4) octoploids produced by

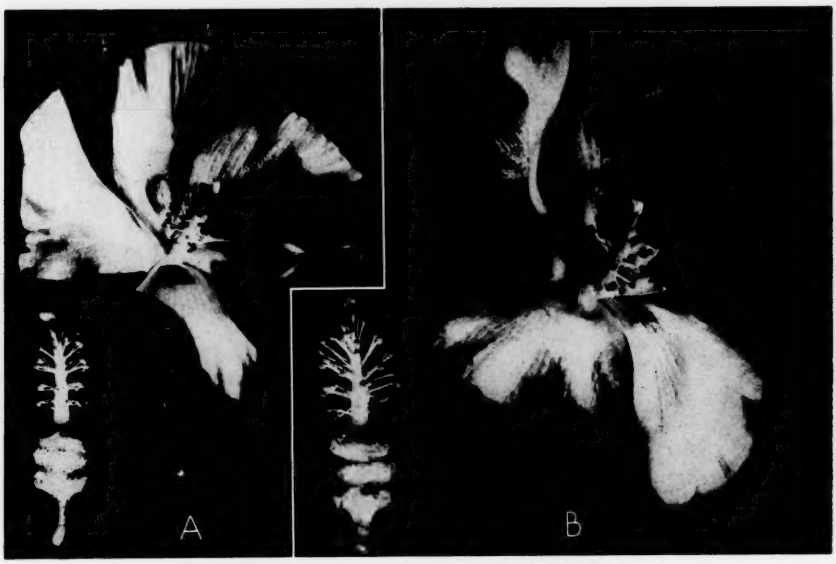
TABLE I. Polyploids of Gossypium Induced with Colchicine

Original Type	Formula of Polyploid*	Percentage Normal Pollen†
F_1 *G. herbaceum*, $2A_1$,* × *G. arboreum* var. *neglectum*, $2A_2$	$2(A_1A_2)$	Probably less than 15
F_1 *G. arboreum* var. *neglectum*, $2A_2$, × *G. thurberi*, $2D_1$	$2(A_2D_1)$	Usually sterile, but 2 fertile flowers have appeared.
F_1 *G. hirsutum*, $2(AD)_1$, × *G. herbaceum*, $2A_1$	$2[(AD)_1A_1]$	65
F_1 *G. hirsutum*, $2(AD)_1$, × *G. arboreum* var. *neglectum*, $2A_2$	$2[(AD)_1A_2]$	85
F_1 *G. barbadense*, $2(AD)_2$, × *G. herbaceum*, $2A_1$	$2[(AD)_2A_1]$	30
F_1 *G. hirsutum*, $2(AD)_1$, × *G. Sturtii*, $2C_1$	$2[(AD)_1C_1]$	85
F_1 *G. hirsutum*, $2(AD)_1$, × *G. harknessii*, $2D_2$	$2[(AD)_1D_2]$	80
F_1 *G. hirsutum*, $2(AD)_1$, × *G. barbadense*, $2(AD)_2$	$2[(AD)_1$-$(AD)_2]$	Most anthers sterile, others few viable grains
G. hirsutum, $2(AD)_1$	$4(AD)_1$	few viable grains
G. hirsutum, $(AD)_1$ (haploid)	$2(AD)_1$	apparently normal
G. barbadense, $(AD)_2$ (haploid)	$2(AD)_2$	apparently normal

*These symbols are explained in a later section of this paper. (Pp. 41-42.)
†Filled with protoplasm and uniform in size and shape.

TABLE II. Hybrids from Induced Polyploids

Formula of Female	Formula of Male	Formula of Hybrid	Species combined in Hybrid
$4A_1$	$2(AD)_1$	$2A_1(AD)_1$	*herbaceum-hirsutum*
$4A_1$	$2(AD)_2$	$2A_1(AD)_2$	*herbaceum-barbadense*
$2(A_2D_1)$	$2(AD)_1$	$A_2D_1(AD)_1$	*arboreum-thurberi-hirsutum*
$2(AD)$	$2(A_1A_2)$	$(AD)_1A_1A_2$	*hirsutum-herbaceum-arboreum*
$2(AD)_1$	$2[(AD)_1A_1]$	$2(AD)_1A_1$	*hirsutum-herbaceum*
$2[(AD)_1D_2]$	$2(AD)_1$	$2(AD)_1D_2$	*hirsutum-harknessii*
$2[(AD)_1(AD)_2]$	$2(AD)_2$	$2(AD)_2(AD)_1$	*barbadense-hirsutum*
$2[(AD)_1(AD)_2]$	$2[(AD)_1C_1]$	$2(AD)_1(AD)_2C_1$	*hirsutum-barbadense-Sturtii*
$2[(AD)_1(AD)_2]$	$2[(AD)_1A_1]$	$2(AD)_1(AD)_2A_1$	*hirsutum-barbadense-herbaceum*



HEXAPLOIDS LARGER THAN TRIPLOIDS

Figure 12

The hybrid between *G. hirsutum* and *G. herbaceum* (*A*), is cytologically a sterile triploid, because *hirsutum*, a 26-chromosome American species, has two differentiated sets of 13 chromosomes. The hybrid may be symbolized as $(AD)_1 A_1$. Doubling the chromosome number with colchicine gives two sets of each of these three groups of 13 chromosomes, or a hexaploid form (*B*) which is fertile. The hexaploids grow and flower like American upland cottons, but have fewer seeds and longer fibers. ($\times$ 0.8.)

doubling the chromosome number in species crosses, (5) octoploids produced by doubling the number in allotetraploid species, and (6) haploids from polyploids.

A system of symbols has been devised, the basis of which is the generally used method of designating each distantly related chromosome set by a letter and repeating the letter to show the number of times a set is present.

Evidence will be presented in a later paper to show that there are apparently at least five distantly related chromosome sets in Gossypium; the sets differ, however, in their degree of relationship. The letter *A* is used to designate the 13-chromosome set in the Asiatic species, *G. herbaceum*, and *G. arboreum;* the letter *B*, the set in the African type *G. anomalum* Wawra. and Peyr.; *C*, the set in the Australian type *G. Sturtii* F. Muell.; *D*, the set in American wild 13-chromosome species, *G. thurberi* Tod.; *G. harknessii* T. S. Brandeg., etc.; *E*, the set in an Arabia-India type, *G. Stocksii* M. Mast. The American 26-chromosome cottons, *G. hirsutum* and *G. barbadense*, etc., according to Skovsted[13], and evidence from the cytological behavior of this type in hybrids with induced polyploids, have the sets (*AD*).

Darlington[3] used the term "differentiated" to describe distantly related sets of chromosomes. "Differentiated" means that given chromosomes, or sets of chromosomes, are unlike in structure, i.e. in arrangement of genes and usually in gene composition, and that the chromosomes are sufficiently unlike that when two of each type are present, the homologous

FLOWER OF A HEXAPLOID

Figure 13

Photograph of a hexaploid produced by crossing a 26-chromosome American upland with a 13-chromosome wild American species. (*G. hirsutum* × *G. harknessii*), and doubling the chromosome number in the resulting sterile hybrid. The hexaploid has about 80 per cent viable pollen; less than half of the ovaries produce normal seeds. (× 0.8.)

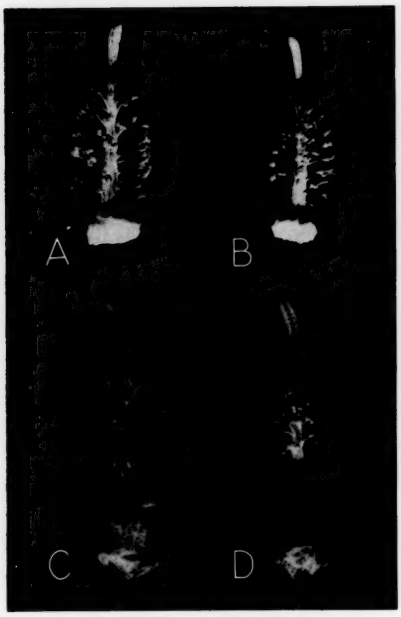
ones usually pair during prophase of meiosis.

In the present system of symbols, well differentiated sets of chromosomes are designated by different letters. Chromosome sets in closely related species with only minor chromosome differentiation are indicated by placing a subscript after the letter designating the set. This is similar to the system used by Kihara and Lilienfeld[7]. For example A_1 designates the chromosome set in one species, and A_2 designates a slightly different set. Thus a formula for a normal diploid may be $2A_1$. The formula for an allotetraploid produced by doubling the chromosome number in a hybrid of two species involving two differentiated sets could be $2(A_1B_1)$. A natural allotetraploid, in which the chromosomes function like the sets in a diploid and in which the basic sets cannot be separated, are indicated by placing parentheses around the symbols. Thus the formula for chromosome sets in a natural allotetraploid might be $2(AB)_1$. A haploid from such an allotetraploid would be $(AB)_1$. The use of formulas of this type is illustrated in Tables I and II.

Characteristics of Induced Polyploids

The autotetraploid $4A_1$ plant from *G. herbaceum*, an Asiatic cultivated cotton, is a typical *gigas* type. Epidermal hairs on leaves and stems are more conspicuous, leaves are about 1.5 times longer and approximately twice as great in surface area, leaves have a deeper green color, internodes appear shorter, and lenticels are more conspicuous than they are in diploids. Stomata size of the cotyledons of an autotetraploid averaged 14 per cent greater in length than those of a sister diploid. Fibers from the tetraploid are 1.4 times longer, and 1.3 times greater in diameter, than those of a sister diploid.

Apparently none of the pollen is functional from the autotetraploid $4A_1$ type, for seeds failed to develop after it was used to pollinate stigmas of flowers from which it came, stigmas of a sister diploid, and stigmas of *G. hirsutum*. A few autotetraploid flowers set seeds after they were pollinated with either *G. hirsutum* or *G. barbadense*, which are $2(AD)$, American cultivated, 26-chromosome cottons. Ordinarily, mature capsules from these crosses contain few seeds, but one had 19 seeds, which approaches the normal number in diploid plants. So far, attempts to produce hybrids of autotetraploid female × the diploid parent have failed. Autopolyploids, $4(AD)$, from American cultivated cottons, *G. hirsutum* and *G. barbadense*, sometimes



ANTHERS OF POLYPLOIDS

Figure 14

The anthers of a fertile hexaploid (*A*) are compared with the smaller anthers of the sterile triploid (*B*). The triploid was produced by crossing 26-chromosome *G. hirsutum* and 13-chromosome *G. sturtii*. Crossing two closely related 26-chromosome species, each containing two distinct sets of 13 chromosomes, *G. hirsutum* × *G. barbadense*, gives a fertile tetraploid. The staminal column from such a flower is shown in *C*. Doubling the chromosome number gives an octoploid with anthers that usually fail to dehisce (*D*). (All 0.8 ×.)

have a few viable pollen grains.

Tetraploids, $2(A_1A_2)$, from F_1 *G. herbaceum* × *G. arboreum* var. *neglectum*, two closely related Asiatic species, have a low percentage of apparently viable pollen.

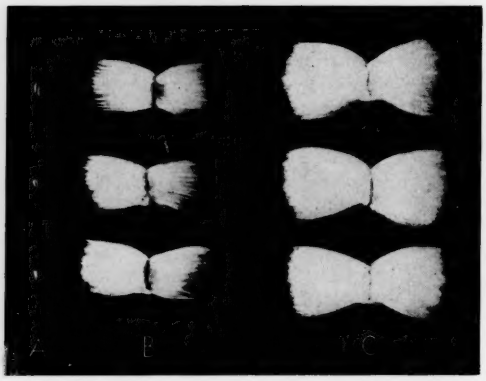
Hexaploids, $2[(AD)_1A_1]$ and $2[(AD)_1A_2]$, produced by doubling the chromosome number in sterile triploids of *G. hirsutum* × *G. herbaceum* and *G. hirsutum* × *G. arboreum* var. *neglectum*, failed to have the pronounced increase in leaf size that is present in the $4A_1$ type. A count of pollen grains in the *hirsutum-herbaceum* hexaploids gave about 65 per cent that appeared normal, while the *hirsutum-arboreum* var. *neglectum* hexaploids had above 85 per cent (Table I). The flowers of the fertile hexaploids are larger, and their anthers are much more conspicuous, than those of the sterile triploid (Figure 12).

These types, at least under greenhouse conditions, have fewer seeds than the better upland varieties. The fiber length of the *hirsutum-herbaceum* hexaploid is about 29 mm., that of the *G. hirsutum* parent 27 mm. and that of *G. herbaceum* 20 mm. These hexaploids grow and flower much like American upland cottons. Flowers of American 26-chromosome cottons usually abort after they are pollinated with *hirsutum-herbaceum* hexaploids; however, the cross was obtained. All $2[(AD)_1A_1]$ flowers pollinated with upland, (AD), pollen have aborted. Flowers of different *hirsutum-herbaceum* hexaploids produced from the same or like triploids had unlike petal spots.

Hexaploids, $2[(AD)_2A_1]$, produced from *G. barbadense* × *G. herbaceum* have only about 30 per cent of apparently viable pollen.

Hexaploids, $2[(AD)_1D_2]$, produced from cultivated American upland 26-chromosome and wild American 13-chromosome species (*G. hirsutum* × *G. harknessii*) have about 80 per cent of apparently viable pollen. (Some of these polyploids, which are assumed to be unbalanced, have a much lower percentage.) A flower of the hexaploid is shown in Figure 13; the sterile triploid flower is similar to the flower shown in Figure 12*A*. Less than half of the ovules in the ovaries of these hexaploids develop into normal seeds. The fibers on these hexaploid seeds are light brown in color, slightly shorter than those on the upland parent, and they are less dense than the fibers on seeds of the upland parent species.

The hexaploids, $2[(AD)_1C_1]$, from a cultivated American and a wild Australian species, produced from the F_1 of *G. hirsutum* × *G. Sturtii*, have about 85 per cent of apparently viable pollen. The difference in the appearance of the staminal column of the fertile hexaploid and



EXTREME LINT-LENGTH DIFFERENCES

Figure 15

The seeds of 13-chromosome *G. Sturtii* (*A*) have such extremely short fibers that they appear to be bare in this photograph. When crossed with a 26-chromosome American species *G. hirsutum*, the resulting hybrids were sterile. The chromosome number was doubled in the hybrids to produce fertile hexaploids (*B*), which have fibers slighty shorter than those of the American parent (*C*).

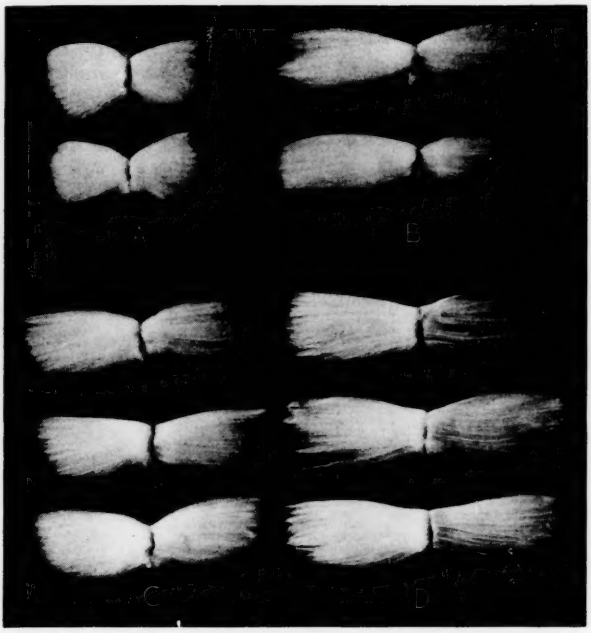
the sterile triploid is shown in Figure 14*A* and *B*. This hexaploid differs greatly from American cultivated cottons, since many of the *G. Sturtii* characters are dominant. The capsules are smaller and the fibers are slightly shorter than those of the American parent. The short fibers were expected since *G. Sturtii* has extremely short fibers (Figure 15). An important point, however, is that the fibers of the hexaploid are smaller in diameter, apparently more uniformly thickened, and have fewer convolutions than those of the American parent. Pollen of this hexaploid will cause capsules of *G. hirsutum* to develop, but the resulting embryos are small and usually non-viable. Crosses of $2[(AD)_1A_1] \times 2[(AD)_1C_1]$ usually abort, but one viable seed was produced.

Doubling the chromosome number in F_1 seedlings of *G. hirsutum* × *G. barbadense* (Pima), two allotetraploid cultivated American species, give octoploids, $2[(AD)_1(AD)_2]$. Some of these polyploids produce a small amount of viable pollen, which will cause seed to set; usually, however, the anthers fail to dehisce (Figure 14*D*). Flowers of these polyploids commonly abort after they are pollinated with either parent species, but crosses were produced. Pollen from the hexaploid types applied to flowers of octoploids will usually cause capsules to develop, although the number of seeds is apparently less than the number in the F_1 with a normal number of chromosomes.

These octoploids seem to have all the hybrid vigor of the normal tetraploid F_1, grow at about the same rate, and form flower buds at the same time. They may, however, require a longer time for flower buds to blossom, and for capsules to mature, than is required in the F_1. Other than in flower characters the most outstanding difference between the octoploids and the normal F_1 tetraploids is the wrinkled and coarser appearance of the octoploid leaves. There is little difference in leaf size, but leaves with double the normal number of chromosomes have a darker green color, and larger epidermal hairs than leaves with the normal number of chromosomes.

Fibers on the octoploid seeds averaged 41 mm. in length, those on the F_1 were 35 mm., those on the *G. hirsutum* parent 22 mm., and those on the *G. barbadense* parent 40 mm. These relationships are shown in Figure 16. The octoploids apparently have an abundance of fibers that are longer than those of the *G. barbadense* parent, which produces a superior type of fiber.

A few observations were made on octoploids produced from *G. hirsutum* × a different variety of *G. barbadense*, namely Sea Island variety Bleak Hall. These octoploids differ from the first ones described in that a larger number of the anthers dehisce viable pollen.



POLYPLOIDS FROM CROSSES BETWEEN UPLAND AND EGYPTIAN COTTON SPECIES

Figure 16

Crosses between two important commercial species of cotton [*G. hirsutum* (*A*) × *G. barbadense* (*B*)] produce fiber of intermediate length in the F_1 hybrid (*C*). Since both parents were natural allotetraploids doubling the chromosome number in the F_1 gave octoploids. The octoploids (*D*) have fibers slightly longer than those of the long fibered parent.

Allotetraploids, $2(A_2D_1)$, produced by doubling the chromosome number in cultivated Asiatic × wild American 13-chromosome species *G. arboreum* var. *neglectum* × *G. thurberi*, are ordinarily male sterile but rarely flowers have viable pollen. They have, however, a high ovule fertility when pollinated with American 26-chromosome, (AD), pollen.

Pure Lines by Doubling the Chromosome Number in Haploids

The occasional appearance of haploid or (AD) plants in Gossypium was first reported by Harland[6]. He found seeds of Sea Island cotton, *G. barbadense*, with twin embryos, and of 16 pairs that reached maturity 14 pairs consisted of one $(AD)_2$ and one $2(AD)_2$ plant. The other two pairs were $2(AD)_2$ plants. Harland also reported two $(AD)_1$ plants in *G. hirsutum*, one from a seed with twin embryos, the other in a field culture. One (AD) plant was found in a strain of *G. hirsutum-barbadense* ancestry. Lehman[9] reported finding a seed with twin embryos in *G. hirsutum*; he found another in 1939. Webber[15] reported finding an $(AD)_2$ plant from a seed with twin embryos. Mr. D. M. Simpson found two seeds of *G. barbadense* with twin embryos in the spring of 1938, from which he grew plants. I saw these plants before they flowered, and their appearance and stomata size indicated that each pair had one haploid, $(AD)_2$, and one normal, $2(AD)_2$, plant.

From a small sample supplied by Mr. Simpson two seeds were found with twin embryos, and an examination of *G. barbadense* (Sea Island) seeds from two other sources gave seven seeds with twin embryos. Of these nine seeds with twin embryos, seven had a combination of one haploid, $(AD)_2$, and one normal embryo; one contained two haploids, and the other had two normal embryos. About 30 additional seeds with twin embryos were found in *G. barbadense* (varieties Seabrook and Bleak Hall). In these varieties Dr. G. F. West found

seeds with twin embryos have a frequency of 1 in 500. The twin embryos were found by germinating seeds on paper towels and examining them when the radicles were 4 to 6 cm. long. Seeds with twin embryos are much rarer in the variety Pima of *G. barbadense*, but one haploid was found.

Early in the summer of 1938 in an inbred line of *G. hirsutum*, var. Superseven, Dr. Thomas Kerr found a plant that was below normal size, with small leaves, and with sterile flowers. It proved to be a haploid. Grafts were made from this $(AD)_1$ plant, and the grafts, together with $(AD)_2$ plants, were treated with colchicine. The chromosome number was doubled in a high percentage of the treated plants, and they have matured seeds. Doubling the chromosome number in the haploids produced genetically pure lines of *G. hirsutum* and *G. barbadense*.

As shown by Skovsted[14], Webber[15], and noted in the present work, chromosome irregularities are comparatively common in $2(AD)$ species. Although there is a possibility that types produced by doubling the chromosome number in haploids have a higher mutation rate than inbred lines, there is little, if any, evidence of it. Satina *et al.*[11] found the mutation rate in haploids of Datura to exceed that of diploids, but diploid plants produced from haploids did not appear to have a higher mutation rate than regular diploids. East[5] reported a high mutation rate in merogonic *Nicotiana rustica* plants, which evidently had their chromosome number doubled in early stages of embryo development, but he believed it to be the normal rate of mutation. Dr. Ernest W. Lindstrom, working with *Lycopersicum esculentum*, failed to find an increased or high mutation rate in diploid plants produced by doubling the chromosome number of haploids.

The differences in rate of mutation (genetic variations) in the different genera may be related to differences in the amount of chromosome irregularities. Polyploids commonly show chromosome irregularities, which may explain the high mutation rate in the allotetraploid *Nicotiana*, and the low mutation rate in the diploid *Lycopersicum*, and *Datura*.

The appearance of an average of 0.2 pair of chromosomes in meiotic metaphase of a haploid, $(AD)_2$, plant, and the presence of 0.14 associations of four chromosomes in $2(AD)$ plants, caused Webber[15] to state that it is impossible to produce pure lines by doubling the chromosome number in (AD) plants. Darlington[3] states, "The high frequency of intra-haploid crossing-over makes it clear that the common notion of a diploid derived from non-reduction in a haploid being necessarily homozygous is a delusion." Darlington's statement is correct in the sense that diploids usually have duplicated genes; however, he is changing the usual definition of homozygous, which means that homologous genes are alike. A pure or homozygous line can be defined as one resulting from the union of identical germ cells. Doubling the chromosome number of haploids gives plants with the equivalent constitution. It can be concluded that doubling the chromosome number of haploids will give pure lines, but the value of such lines for genetic and breeding work will depend upon their rate of mutation.

Discussion

Although many more polyploids can be induced in Gossypium, the ones that have already been obtained represent several significant types, since they were produced in the following cottons: 1, cultivated American and Asiatic species; 2, hybrids of the two cultivated 26-chromosome American species; 3, hybrids of cultivated 13-chromosome Asiatic species; 4, hybrids of cultivated American 26- and Asiatic 13-chromosome species; 5, hybrids of cultivated American 26- and wild American 13-chromosome species; 6, hybrids of American 26- and Australian 13-chromosome species; and 7, hybrids of cultivated Asiatic 13- and wild American 13-chromosome species.

One point of caution in producing polyploids in Gossypium with colchicine is that polyploids produced from the same stock are in some cases unlike. For example, octoploids from F_1 *G. hir-*

sutum × *G. barbadense* differ in fertility, and hexaploids of *G. hirsutum* × *G herbaceum* differ in petal spot. These differences are probably explained by the report of Bergner, *et al.*[1] that polyploids produced with colchicine sometimes have plus or minus one or more chromosomes than the regular polyploid number. This makes it advisable to produce several polyploids from the same stock.

The polyploids thus far induced in Gossypium show some of the limitations of polyploidy in plant breeding and specifically in the breeding of cotton.

Polyploids that were produced from pure Gossypium species, *hirsutum* and *herbaceum*, are pollen sterile; polyploids from crosses between closely related species have a high percentage of pollen sterility; polyploids from species of intermediate relationship have intermediate sterility. Surprisingly, polyploids of one combination of distantly related species usually are pollen sterile, although flowers with fertile pollen have appeared. Polyploids showing partial sterility are expected to segregate; and, although polyploids from hybrids of distantly related species may have a high fertility, they may have other characteristics that prevent them from being suitable for cultivation.

In spite of the limitations, there are some promising possibilities. Initial polyploids that are undesirable for cultivation probably can be crossed back to the cultivated parent species, and genes from one species can be transferred to another.

In regard to partial sterility, the work of Kostoff[8] is encouraging. He selected in five generations stocks with normal fertility from a polyploid of tobacco showing about 50 per cent sterility. In order for most Gossypium polyploids to be of commercial value one thing that will be necessary will be to produce types with a small percentage of sterility from initial polyploids having 15 to 50 per cent sterility.

Another promising possibility is illustrated by the *hirsutum-herbaceum* and the *hirsutum-arboreum* var. *neglectum* hexaploids from hybrids between cultivated American and Asiatic cottons. The chromosome structural relationships between the species in each cross are almost the same, but the *hirsutum-herbaceum* hexaploid has only about 65 per cent of apparently viable pollen whereas the *hirsutum-arboreum* hexaploids have above 85 per cent. This seems to indicate that if a number of polyploids of different combinations can be produced from two general types that the various combinations might give polyploids that differ greatly in fertility and other characteristics.

Crosses between distantly related types may give polyploids that breed true. Genes from wild species, when combined into polyploids with cultivated types, produce new types of fibers, and certain genes doubtless will influence disease and insect resistance and growth habits.

Summary

Polyploids were produced from eleven types of Gossypium. The induced polyploids include auto- and allotetraploids, hexaploids, and octoploids, and hybrids were made that gave pentaploids and heptaploids. The polyploids were produced by the immersion of apical meristems in 0.2 per cent aqueous solution of colchicine for 24 hours. This treatment caused 10 to 55 per cent of the treated plants to produce polyploid branches.

A system is outlined for writing formulas that make the composition of polyploids clear. Each chromosome set is given a capital letter and a number is placed before the letter to indicate the number of times a chromosome set is present.

Polyploids from sterile hybrids between distantly related types are usually fertile, but one exception was found. Polyploids of pure Gossypium species or of hybrids between closely related species are female fertile, but are almost or completely male sterile. Fibers on induced polyploids usually have a greater length and diameter than those of the types from which they came.

Pure lines of *G. hirsutum* and *G. barbadense* were produced by doubling the chromosome number of haploids.

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Gory Details Galore

TWO outstanding reviews of the field of the Blood Groups have appeared during the current year, the first of which, by W. C. Boyd, has already been reviewed in this Journal (August 1939). The other is a drastically revised edition of A. S. Wiener's "Blood Groups and Blood Transfusion," (C. C. Thomas, Springfield, Ohio, 1932).

The work by Boyd is more concise, soft pedals such topics as forensic applications and hospital techniques but reproduces numerous detailed tables of great utility to the laboratory scientist.

In contrast to Boyd, Weiner deals more with the practical applications. He treats such topics as the blood group specifications, including *A*, A_2, *B*, *M*, *N*, *P*, *Q*, *X*, cold agglutinins, group specificity of body fluids and their heredity. The book considers indications for transfusion, anthropological and mammalian distribution. Then he gives details of the use of blood groups in disputed parentage suits, mixed baby controversies and for personal identification in murder cases. Under 20 chapter headings, the medical man, the anthropologist, the serologist, the lawyer, the geneticist, and the practicion, will find a well balanced account applying to his specialty.

Wiener's volume is the better for classroom assignment, but students of the blood groups will want both books.

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